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Supreme Court, U.S.
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CLERK

No.

# In the Supreme Court of the United States

**OCTOBER TERM, 1988**

ALCOLAC, INC.,
*Petitioner,*

vs.

BETTY ELAM, et al.,
*Respondent.*

**Volume I**
**APPENDIX TO**
**PETITION FOR A WRIT OF CERTIORARI TO THE**
**MISSOURI COURT OF APPEALS,**
**WESTERN DISTRICT**

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IN THE MISSOURI COURT OF APPEALS

WESTERN DISTRICT

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|---|---|---|
| BETTY L. ELAM, CLARENCE ELAM and LINDA LOU SANDERS, | ) | |
| ETHEL M. BERRY, | ) | |
| CARL C.* and JACQUELINE BERRY, | ) | |
| VIRGIL and DOROTHY BRADLEY, | ) | |
| EDWARD and MALVA GEHLKEN, | ) | |
| DAINIE and MARY LUCETTA LANDON,* | ) | |
| GWENDOLYN and JOHN C. LAWRENCE, | ) | |
| GLEN A. and BERNICE MILLER | ) | No. WD 38,105<br>CONSOLIDATED |
| CHARLOTTE A. and JOHN PHILLIPS, | ) | Opinion Filed:<br>November 1, 1988 |
| DANIEL CHARLES and JOYCE PRYOR and AMBER CROSS, | ) | |
| ARNOLD L., JOY R., JOYCE and TAMMY SOMMERS, | ) | |
| JAMES R., KAY D., WILLIAM LANCE, LISA and LYLE TURLEY, | ) | |
| GENEVIEVE and RALPH H. WITHERS, | ) | |
| Respondents-Appellants, | ) | |
| vs. | ) | |
| ALCOLAC, INC., | ) | |
| Appellant-Respondent. | ) | |

APPEAL FROM THE CIRCUIT COURT OF JACKSON COUNTY

The Honorable Tom J. Helms, Judge

Before Shangler, P.J., Manford and Nugent, JJ.

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*Died during pendency of appeal and the spouse was substituted as party by order.

ELAM, et al. v. ALCOLAC, INC.

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This suit involves thirteen actions brought by thirty-two plaintiffs against Alcolac, Inc. and plant manager Fischer for injury to their persons and property from toxic spills and emissions from a chemical facility in Sedalia, Missouri. The actions, consolidated by order of the trial court, were in four counts and each sought recovery of compensatory and punitive damages. Only two counts were submitted to the jury: One, for the diminished market value of the residences, sounded in nuisance. The other, for injury to the persons, sounded in negligence. The jury, after a trial of more than four months, returned verdicts against Alcolac and awarded to each plaintiff on the claims for personal injury, $200,000 as compensatory damages and $1,387,096.70 as punitive damages. The award for property damage varied as to each claim. Alcolac thereafter moved for a judgment notwithstanding the verdict or, alternatively, for a new trial. The trial court denied judgment n.o.v., but granted a new trial as to damages only as to each of the thirty-one[1] personal injury verdicts.

The several plaintiffs appeal the order to set aside the awards for personal injury damages on the negligence count and the grant of a new trial on those issues. Alcolac appeals the denial of judgment notwithstanding the verdict on both negligence and nuisance counts, the entry of judgment for property damage on the nuisance count, and the entry of judgment of liability on the negligence count.

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[1] The motion for directed verdict by defendant Fischer was sustained as to all the plaintiffs, and the motion for directed verdict by defendant Alcolac was sustained as to the plaintiff Edward Gehlken. Thus the causes of action of the thirty-one plaintiffs who remained were submitted against Alcolac only.

## PART ONE

### The Litigation History

Alcolac commenced its Sedalia operations in May of 1978. In July of 1979 five of the thirteen plaintiff families filed suit in Pettis County with complaints of injury from the Alcolac operations. The five suits were voluntarily dismissed in February of 1981. In December of 1981 the suits were refiled in Jackson County, other suits were commenced, and eventually all were consolidated for trial. Alcolac made answer to the petitions. Alcolac also pleaded a multiple counterclaim. Count I was for Abuse of Process: that the plaintiffs and others in concert maliciously schemed to harass and force Alcolac to cease operations at the Pettis County plant, and to that end commenced an unfounded suit against Alcolac in Pettis County, took no initiative to advance the litigation and later dismissed the suit, but resumed the litigation in Jackson County - not for the redress or remedy of any civil wrong, but for ulterior purposes. Count II was for Civil Conspiracy: that the acts alleged against the plaintiffs in Count I constituted a civil conspiracy. Count III was in Prima Facie Tort: that the acts of the plaintiffs were intentionally done with the purpose to injure Alcolac, and without justification. The counterclaim sought recovery for compensatory and punitive damages as to each cause of action. Alcolac attempted proof of the cause of action through the cross-examination of the several plaintiffs, but without avail. At the conclusion of all the evidence Alcolac dismissed the counterclaim against all the plaintiffs with prejudice. Accordingly, the appeal and cross-appeal are from a final judgment and engage our jurisdiction to review.

What remained was a massive litigation of thirty-one separate causes to action, joined for adjudication through the amenity of consolidation, but tried, submitted and determined by the jury as separate suits. It was a litigation which engaged the jury from August 12, 1985 to December 23, 1985 [four and one-half months] presented one hundred and sixty-five witnesses as well as hundreds of exhibits, and is recorded in ten thousand pages of transcript. The counter-appeals contest not only the validity of the submissions and the verdicts as returned, but also the judgment of the court which validates the proof of the causes of action but nullifies the money amounts returned by each verdict for personal injury and punitive damages. Our review entails the determination of the submissions, the validity of the verdicts, and the propriety of the judgments entered by the court - among the other claims of error.

II

A

The Alcolac Plant: Its Design and Operation

Alcolac, Inc. manufactures specialty chemical products for industry and commenced such operations in 1950 in the city of Baltimore. It is now an international company. The special chemicals manufactured by Alcolac include surfactants and functional monomers. Surfactants are surface active agents or soaps used in cosmetics and a range of other products. Functional monomers are building-block chemicals used to prepare polymers for adhesives, plastics, textiles, and such. The Alcolac management recognized the dangers to the environment from such production activity and so launched WAPORA, an environmental management

subsidiary, which sells advice on pollution control to industry as well as government.

Alcolac then sought a site in the Midwest for an additional facility, and selected an area contiguous to the city of Sedalia. To deal with the environmental concomitant of the planned facility in Sedalia, in 1975 Dr. Vsevolod Blinoff, founder and chairman of Alcolac, designated the top executives of the company as an ad hoc committee. They included Charles Anderson, Alcolac president, Peter Bouroff, Alcolac vice-president, Dr. Jacob Bregman, president of WAPORA, and Blinoff, himself. It befell Bregman and WAPORA to design the environmental control system for the Sedalia facility. The design Bregman and Blinoff envisioned was a model of environmental safety - a plant [as described by Bregman] "that could make products that were toxic and hazardous and yet keep them in such a manner that the environment would not be affected." It was the specific Blinoff instruction that Bregman and WAPORA design and construct "a zero discharge plant" - a state of the art model. Anderson, president of Alcolac and an accountant by training [described by Bregman as "a bottom line kind of guy"], opposed many of the environmental controls as too costly and "a lot of baloney." The design was drawn and construction began nevertheless.

The composite facility included three production buildings: a building for the manufacture of surfactants or soaps, another for the manufacture of monomers, and an environmental control building.[2] The production from both the surfactant and monomer plants resulted in wastes, both in liquid and vapor form. It was the design of the pollution control processes at the plant to remove the organic chemical contaminants from the wastes, and

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[2]See Appendix A. The Alcolac facility also included four biodegradation ponds, whose function the opinion describes, as well as a warehouse and lakes.

to expunge their odors, before emission. The design provided for carbon filters and scrubbers to remove the contaminants and odors from the wastes, incinerators to consume the organic matter removed from the liquids and vapors, and pipe lines to emit the liquids and stacks to emit the vapors. It was the theory of the design that the pollution control processes would render the otherwise toxic and odorous wastes harmless to the environment.

To dispose of the liquid wastes, underground pipes ran from each of the three structures to four bio-oxidation ponds. There was also an underground pipeline between the monomer plant and the environmental control building. It was the function of the bio-oxidation ponds to degrade the contaminated remnants discharged into them through the conduits from the surfactant building and the environmental control building. The bio-oxidation ponds were designed on the same principle as any municipal sewage treatment lagoon: the ponds were filled with plain water and the bacteria allowed to develop; as the wastewaters [theoretically scrubbed free and filtered of contaminants] are gradually introduced, the bacteria degrade them. Thus, the design, as applied to both the surfactant and the monomer wastes, intends that the organic chemical contaminants be removed before discharge into the bioponds.

In the case of the soap plant, the wastes were to be treated by acid scrubbers to remove the chemical residues and odors from the liquid and vapors. The liquid was then effused into the biopond and the waste vapors, then also cleansed of any acid mist, were emitted through the surfactant stack.

In the case of the monomer plant - where the toxic chemicals were used for production - one liquid waste stream flowed into the environmental control building and over carbon filters for the removal of the organic materials before discharge into the biopond for biodegredation. The other liquid waste,

suspensions of undissolved organic particulates, was piped into the liquid incinerator for burning, then through scrubbers to clean the odors, before emission into the atmosphere as vapor. The toxic vapor wastes from the reactors in the monomer production process were filtered through a system of carbon beds in the monomer building and the organic residues burned off in the "fume incinerator" before emission.[3]

The design intended, therefore, that the liquid emerge from both the surfactant and monomer plants free of organic matter and then flow into the bioponds as uncontaminated wastewater. It was the theory of the design that the wastewater then biodegrade through a series of four bio-oxidation ponds, each of which retained the wastewater for thirty days before it flowed by gravity into the next pond, and finally collected [after the 120-day cycle] into a sedimentation pond for reuse - either for field irrigation or as washwater for the plant reactors.

This system of controls notwithstanding, both the monomer and environmental control buildings were designed with valves meant to be unlocked only for the release of wash water and storm water to the bioponds. Instead, operators in the

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[3] The WAPORA design for the Alcolac plant at Sedalia selected the carbon adsorption system for the control of the pollutants from the monomer production process as the ideally efficient alternative. It also proposed "scrubbers" to cleanse the liquid waste from the monomer production, but only "[i]f operational testing shows that a potential environmental problem remains after these measures are taken." It became evident to the Missouri Department of Natural Resources [the official agency with oversight of the Alcolac operations] that the system for pollution control, as installed and operated, was not effective to meet environmental control standards. It was a condition of the abatement agreement concluded by Alcolac and the Department of Natural Resources on May 30, 1980 [as our discussion more fully delineates] that Alcolac install two alkaline scrubbers to remove the acrylates from the liquid waste streams and the gaseous waste streams from the monomer reactors. They were duly installed by Alcolac.

monomer and environmental control buildings at times used the valves to bypass the waste treatment features of the facilities and pipe raw, untreated toxic chemical wastes directly into the open air bio-oxidation ponds outside. Also, the wastewater from the monomer process was intended for discharge into biopond one, and that from the surfactant process into biopond two - [presumably, because the soap wastewater was more readily biodegraded than the monomer wastewater]. Notwithstanding, it was the recurrent practice to emit both the soap wastewater as well as the untreated monomer wastewater into biopond one, with the result that the toxic monomer wastewater intermixed with the soap wastewater and was borne off the Alcolac premises as puffs of foam. Notwithstanding also the sense of the design that the liquid incinerator function as the means of disposal of particulate contaminant wastes from the monomer process, the device malfunctioned from the outset of Alcolac operations and, in mid-1981, was discarded as a means for the disposal of toxic waste.

B

## STARTUP OF OPERATIONS 1978

Alcolac operates under license of the Missouri Department of Natural Resources [DNR]. That agency has the primary responsibility to enforce environmental compliance. Alcolac is also monitored by the Environmental Protection Agency [EPA] and the Occupational Safety and Health Administrations [OSHA].

Alcolac commenced operations in the Sedalia plant in May of 1978. Some months earlier, in November of 1977, Bregman had circulated to Blinoff, Anderson, Bouroff and Fischer [then plant manager], a memorandum of "comments on the Sedalia plant start-up." Bregman had recently visited the facility and noted a

number of problems he wanted to be certain were corrected before the onset of production. He explained: "They have to do with pollution control systems, and very heavy emphasis on training the plant personnel." The memorandum recommended, among others, that all the pollution control equipment required for proper operation of the plant be installed and tested before the "first batch" of production; that all plant personnel "go through an intense training session on the use of the pollution control manual" [the Environmental Control Systems Operating Manual devised for the plant by WAPORA]; that "[a] system of very close checking of the bio-oxidation ponds for odors" be established to ensure that "no noticeable odors from them get anywhere near the property lines or the Alcolac building." These goals, Bregman concluded, were never met.

Alcolac began production nevertheless some six months later, and almost from the start the liquid incinerator and the bio-oxidation pond systems were sources of complaint. The incinerator emitted vapors and hazes over the environs and the bio-oxidation ponds emitted odors. Alcolac called on WAPORA to allay these problems. Bregman assigned to David Boies, the engineer who had designed the Alcolac plant, the liquid incinerator concern, and to engineer Dr. Leroy Reid, an expert in the treatment of toxic waste, the bio-oxidation ponds odor concern. Boies and Reid concluded the assignments and reported to Bregman.

Reid completed the first phase of the assignment in July of 1978 and in a memorandum expressed concern that the carbon filter system in the environmental control building was used to overload with the consequence that toxic wastes from the monomer process were allowed to enter the bio-oxidation ponds. A month later Reid resumed the investigation and found the bio-oxidation ponds still toxic as well as odorous. Reid also discovered on that second visit that the drain valves which lead

from the monomer building to the bioponds were not locked closed as normal procedure intended. This allowed chemical spills in the monomer building to flow directly into the waste pipes of the environmental control building for discharge into the biopond without any prior cleansing in the liquid incinerator. Dr. Reid not only reported to Alcolac that the ponds were being contaminated with toxic wastes, but that the toxic chemicals lighter than water could attach themselves to the foam created by the aerators in the bioponds and be blown in the air or become vapor - and so present a hazard to the environment. Dr. Reid also recommended that an additional aerator be installed in the bioponds to infuse the water with more oxygen, and so dispel the odors. Reid also reported to WAPORA, by a later memorandum of October 31, 1978, with copies to Anderson and Bouroff at Alcolac, that the chemical data collected indicated that the biopond was receiving loadings of over 18 times design conditions.

Reid continued the investigations into 1979. The increments of chemical wastes from the increased production outstripped the capacity of the bio-ponds to degrade the chemicals so that a sludge accumulated at the bottom of the lagoons. Odors were emitted of the "rotten egg type, which were probably from hydrogen sulfide." On several occasions Reid experienced "a burning type sensation to the odor coming off the lagoons" to the membranes of the nose. His reports noted also that the acid scrubbers in the surfactant building were not as effective as expected so that soap flowed into the bio-oxidation ponds and put an additional stress on a system already overburdened. Other memoranda reported to Alcolac and plant manager Fischer that the employee who operated in the environmental control building was untrained, and not "capable of doing a proper job." A memorandum to Anderson at Alcolac in June of 1979 reported other problems: the rupture of a disk [a monomer reactor safety device] which

sprayed toxic chemicals into the air. That memorandum also reported that a drain outside the environmental control building discharged untreated chemicals into a ditch and from thence into the atmosphere - among other numerous erratic environmental practices noted as to Alcolac. Reid reported also that strainers on the pumps in the monomer building clogged, so that the pits overfilled with the consequence that the raw untreated chemical waste flowed directly to the bio-oxidation ponds, still toxic. Reid noted also on the occasion of another visit to the Alcolac plant a spill of dodecylbenzene sulfonic acid at the surfactant plant. He commented also that the environmental technician [LeMaire] was untrained. He noted that the position was very important "as any data he produces must be valid" and urged that LeMaire be trained. Reid urged also that the employees be impressed with the requirement for good environmental control measures. He observed that the plant lacked a "spill response program" - a team trained and equipped to handle spills of corrosive and hazardous chemicals in order to prevent damage to personnel and the environment.

In August of 1979, the Reid memorandum cited evidence of several major chemical spills - which "probably violate either or both Federal or State law." He commented, "[b]oth were serious and major spills which could pollute either streams or groundwater acquifers." That memorandum concluded with the summary: "There seems to be new problems developing at the plant each time I visit," and urged management "to take a highly active role in enforcing the proper conduct of employees to reduce danger to the personnel health and to the environment." It concluded: "Haphazard handling of chemicals should not be tolerated."

Reid submitted numerous recommendations to Anderson and the other Alcolac officials on solutions to the bioponds pollution problems - with specifications, design calculations and cost estimates - but every recommendation met resistance or delay in implementation. In addition to the memoranda, Reid warned the Alcolac officials that the environmental problems would exacerbate. The major concern, as he saw it, was that the chemical waste from the increase in the production schedule "would overcome [the improvement to the bioponds] we had planned before it even got installed." The ultimate resistance to the recommended solutions to the bioponds pollution problem was an economic resistance "clear at the top" - Alcolac president Anderson. Reid had the impression that Alcolac either did not have the money to spend or did not want to spend the money.

Boies also pursued his assignment of June, 1978, to investigate the liquid incinerator and also reported to Bregman. Boies died in 1980, five years before the trial, so Bregman related the results of that phase of the investigation. In the course of the investigation in June of 1978, Boies paid a visit to the woman who complained that the liquid incinerator caused vapor and smoke to drift over her home, and Boies made apology. Boies composed a detailed memorandum about that incident, gave it to Bregman, and sent a copy to Alcolac. Anderson, irate that Boies should have apologized, demanded that "Boies be pulled off the project and [not] to be put on it anymore." Blinoff, the founder of Alcolac and champion of "a zero discharge plant" had died by then, and Anderson succeeded him as the dominant figure in Alcolac. Thus, Bregman acceded to the Anderson demand, and removed Boies from the Sedalia project.

Boies had written other memoranda concerning his investigation of the Sedalia project, but they were missing from the WAPORA files. Bregman still retained a related document, however

- a typewritten draft of a proposed memorandum to Alcolac prepared by Boies and submitted to Bregman, who inscribed his comments. The memorandum in final form - circulated to Anderson and other Alcolac officials - was also missing from the WAPORA files, but Bregman reconstructed its content from the document at hand. The memorandum was a catalog of "things that were remaining to be done" to bring the pollution control systems to proper function. One item commented: "No one person responsible for pollution control." It meant, Bregman explained, that "Alcolac did not have a trained competent person who looked at the pollution control systems, made sure they operated right." Another item commented that the "sampling points on the vent absorber units" were not yet installed, and hence "whether stuff was escaping into the air or not and how much or what" could not be determined. Alcolac always responded "they were too busy running the plant to do this." Another item commented that the major problem as to water pollution control was that "a much higher organic load than had been predicted" was emitted into the bio-oxidation ponds. That is to say, the production of organic compounds at Alcolac exceeded the design level based upon the production prediction provided by Dr. Bouroff. The result was that a much higher organic load wound up in the oxidation ponds than allowed for by the design. Another item commented that the Alcolac employees were not using the record forms supplied by WAPORA to monitor the bio-oxidation ponds.

Another "major basic problem" the Boies memorandum mentioned was from the operation of the liquid incinerator, which generated haze in the atmosphere. Bregman explained that the local residents were restive about that condition of pollution, and the newspaper reported that uneasiness. Bregman assured the citizenry at a meeting that "these things were going to be solved." That condition apparently came to the attention of the

Department of Natural Resources, and the Alcolac officials, Anderson and plant manager Fischer, in the company of Bregman met with the agency in Jefferson City. Bregman and the others disclosed to DNR the plans for air pollution control, and the agency was satisfied. Anderson informed WAPORA shortly after that meeting that Alcolac would "handle all future dealings with the State of Missouri" without "involvement" of WAPORA. The eventual consequence rather was to remove WAPORA as consultant altogether. In the interim, however, Bregman - as requested by Alcolac - met with yet another citizens' group, an environmental committee, to allay the concerns of the Sedalia citizenry and to assure them that the air pollution problems would be resolved. Bregman then still believed that the recommendations of the Boies memorandum, as well as his own, would be implemented. Anderson refused them, however, because: "[I]t cost too much money."

Alcolac discharged WAPORA from the Sedalia project in December of 1979. A month or so before, Bregman dispatched a last memorandum to Alcolac president Anderson and vice-president Bouroff, among others. A copy of that memorandum was also missing from the WAPORA files. Bregman, however, retained a sketch, written in his personal hand, from which the formal memorandum was composed. It was a summary of past operational problems at Sedalia, the present state of those problems, and recommended solutions. The memorandum noted once again, as before, the need for a strong and experienced engineer for the maintenance of the expensive and complex pollution control equipment, that the employees be trained in odor detection and control, and that the standard operating procedure delineated in the Environmental Control Systems Operating Manual composed by Wapora for the Sedalia plant be observed - all recommendations already made to Alcolac by WAPORA but not implemented. The memorandum also

informed Alcolac, once again, that the monomer plant was producing three times the chemical waste the design capacity of the bio-oxidation ponds allowed. Bregman reported also that the odors were caused by the "sloppy handling" of the chemicals used in the monomer process and could pose a considerable toxic control problem if not handled correctly. It was communicated to Alcolac, also, that a number of the chemicals used in the monomer production had "significant air pollution potential" so that the plant environmental control system needed to be flexible and reliable. Among the chemicals specifically mentioned as environmental hazards were allyl alcohol, dimethyl sulfate, epichlorohydrin, ethyl acrylate, methyl methacrylate and toluene sulfonic acid.

Bregman testified that Anderson refused to implement the recommendations of the last memorandum, as he had the predecessors, as too costly. Bregman deemed the environmental system designed for Alcolac worthy of a national prize, and told Anderson that "if he would fix this up, we would have the best one going" - but to no avail. In December of 1979, Alcolac president Anderson removed WAPORA from the Sedalia project. Bregman cited - as of that date of departure - the continued reluctance by Anderson to spend the funds necessary for these essential pollution controls: $150,000 to install hydrocarbon monitors; another $100,000 or so for an aerator system to facilitate the oxidation of the excess chemical wastes in the ponds due to unforeseen increased production, and about $500,000 to $1,000,000 to redo the liquid incinerator. Bregman reiterated as the "single-most important factor" to a proper system of environmental control "a highly qualified environmental engineer to make sure that the systems work and to give the fellow the authority to make corrections when they don't work." It was a recommendation Anderson

"strongly opposed," because of the salary entailed, and because the "corrections" would involve "an ongoing expense."

Some two years later, after the commencement of suit against Alcolac, Bregman heard from Anderson once again. As Bregman related the incident, Anderson said that "there were some kooks who filed a lawsuit" and he asked Bregman "to destroy the WAPORA project file on the Sedalia project." Bregman refused the request as "unethical, probably illegal and certainly unprofessional." Of the two files at WAPORA on the Sedalia project, one disappeared altogether.

Alcolac president Anderson was called by the plaintiffs and was examined as an adverse witness. Anderson was a member of the ad hoc committee appointed by Alcolac founder and Chairman, Dr. Vsevolod Blinoff, and so was involved in the decisions on the design and construction of the Sedalia plant - the environmental control system, included. Anderson acknowledged that as early as January of 1978, he was aware the Sedalia production process involved many dangerous and highly toxic chemicals. He acknowledged also the numerous WAPORA reports and recommendations, written and spoken, during 1978 and 1979 [the first two years of Sedalia operations] concerning the improvement of the environmental control systems and that Alcolac was not willing to spend money to implement them all.

They did [according to Anderson] use the Environmental Control Systems Operating Manual prepared by WAPORA at the Sedalia facility.[4] Alcolac did purchase one of the eleven hydrocarbon systems recommended by WAPORA to monitor chemical

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[4] There was some intimation in the Bregman testimony, also, that Alcolac installed some sort of aeration system in the biopond, but apparently not of a design WAPORA considered effectual to degrade the volume of additional toxic chemical waste from the increased production.

emissions. That monitor did not function, however, and Alcolac depended on periodic samples taken from the stacks and dispatched to independent testers - "several times over a couple of years" - in lieu of the analytic monitors WAPORA found essential to detect, warn of and control the toxic emissions. The liquid incinerator had not functioned effectively from the outset of production so that in May of 1980 [after the dismission of WAPORA] Alcolac vice-president Bouroff was prompted to report to president Anderson among the items of "Progress As Of May 1980":

> Items 1 & 2: Our liquid incinerator has the poorest performance ever seen. Most of the time it is out of commission due to all kinds of mechanical problems. It is an absolute waste of time and money. We consider it a 100% failure and the most costly operation. Also, part of our environmental problems were/are caused by this piece of equipment.[5]

The malfunction notwithstanding, Alcolac continued to use the incinerator as the mode to dispose of the liquid waste. The result was that the waste was only partially incinerated, if at all, so that the residue was vented into the atmosphere still as toxic, or even more so, than before. In 1981, the use of the liquid incinerator was discontinued altogether. The hazardous waste intended for disposal by that method was hauled away thereafter.

Anderson recalled that during one of his periodic sojourns at the Sedalia plant - in 1978 or 1979 - he had occasion to visit Charlotte Phillips [an eventual plaintiff] at her home north of the facility to discuss a complaint of odors. He never knew of any incidents of odors from Alcolac "going beyond the property boundaries" and contested her testimony that on the occasion of that visit the odors were so pronounced "that everyone's eyes were watering and people were choking," including

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[5] See APPENDIX F for the full text of the document

Anderson, himself. Nor had Anderson ever seen foam from the bio-oxidation ponds "blow beyond the bounds of the Alcolac property" except for a photograph in evidence which depicted such an accumulation beyond the bournes of Alcolac. He conceded, however, that "[w]e have been cited by the DNR [Department of Natural Resources] for odors that have gone off of our property before." The numerous complaints of odors from Alcolac made by neighbors of the plant, he said, were personally investigated and never corroborated. The complaints of odors, Anderson said, were unfounded. Anderson conceded that he was advised that the bio-ponds were regularly being overloaded, but on one occasion only, and then by Dr. Reid. He acknowledged that Alcolac employee Joseph LeMaire was the person Dr. Reid alluded to in his 1979 report as the environmental control technician totally untrained and incompetent for such a position. Anderson was adamant, however, despite even the testimony of LeMaire to the contrary, that LeMaire functiones as a maintenance worker - not an environmental control technician. Anderson acknowledged that Alcolac officials had met with the DNR some ten times since 1978 concerning the emission and odor problems at the Sedalia facility. He denied any overture to Bregman to destroy the WAPORA files consequent to the initiation of suit by these plaintiffs.

Joseph LeMaire was employed at Alcolac between August of 1977 and January of 1984. His testimony as to his employment function and the regularity of the plant operations was in essential contradiction to that given by Alcolac president Anderson. His service with Alcolac, therefore, encompassed the entire period of the WAPORA presence there - and beyond. LeMaire came to Alcolac at the age of eighteen years as a maintenance worker and groundskeeper. He had dropped out of school at the 10th grade, but later gained the General Equivalency Diploma. That was the

extent of his formal education. In the summer of 1979, the employee in charge of the environmental control building was discharged for drunkenness on the job, and [according to LeMaire] without training or even the guidance of an operation manual, LeMaire was placed in charge of environmental control. He was accorded the title: "environmental control technician." LeMaire described the process by which the environmental control building devices were designed to function: the liquid waste stream from the monomer plant [where the most toxic chemicals were used] was conducted into a sump pit in the environmental building and from there was filtered over charcoal Calgon units and the liquid, as treated, was then discharged into biopond one.

LeMaire described episodes of repeated chemical spills at the plant. Toxic spills were often "push[ed] down the drain with squeegees." The valves which controlled the monomer drains were kept open as a matter of course so that the spilled toxic liquid flowed directly into the environmental control sump pit and was allowed to discharge into the biopond without completion of the charcoal decontamination process. Also, on occasion, the environmental control drains would become so overloaded that the waste liquid backed up, and the waste was routed around the environmental control filtration system, still toxic, directly into the biopond. On other occasions, the toxic liquid overflow was simply allowed to drain from the environmental control building to the outdoors, and from thence into Little Shaver Creek. LeMaire could detect the odors of toluene and allyl methacrylate, among other toxic compounds, from these "washdown waters" from the monomer building. LeMaire also described occasional dispersions of chemicals into the atmosphere from explosions in the monomer plant [a phenomenom noticed by the WAPORA report of Dr. Reid to Alcolac president Anderson]. The exposure to these toxic wastes burned the eyes, irritated the throat and impaired the breathing.

LeMaire described the odors emitted by biopond one as of "rotten eggs or a bad sewer system," or sometimes, "a sweet smell, or a sour, bitter, smell," depending on which untreated chemical wastes were in the pond.

The "washdown from the soap [surfactant] plant" also collected in bio-oxidation pond one. Thus, when the chemical wastes from the monomer building were released directly into the pond uncleansed by filtration, they mixed in that toxic state with the suds from the soap building. Those suds, LeMaire observed, left the Alcolac premises "like a snowstorm" whenever the wind blew, and at times built the suds to a height of "close to 15, 20 feet tall." Lemaire and the other workers, accoutered in protective gear, attempted to control these occurrences with sprays or other such substances.

LeMaire recounted that during his service at the Alcolac plant, the Department of Natural Resources made periodic inspections. Alcolac received advance notice of the prospective inspections and "cleanup parties" were held to prepare the plant for the inspectors. The agency discontinued the inspections when the plaintiffs commenced these lawsuits, and then "the cleanup parties stopped." LeMaire was terminated from employment in January of 1984 shortly after he suggested to Alcolac president Anderson and vice-president Bouroff that the Missouri Department of Conservation be summoned to investigate the death of numerous fish in a pond which adjoined the plant. Anderson did not recall the episode, although he acknowledged LeMaire was fired. It was the installation of a twenty-year-old with no training [LeMaire] as environmental control technician which prompted Reid to comment in a WAPORA memorandum to Alcolac that the appointment "made a joke of [the WAPORA] recommendations."

Paul Faulconer worked at the Alcolac plant and confirmed, and enlarged on, the operational malfunctions described by LeMaire. He worked there for six months from April of 1979, and so his service was during the WAPORA presence at the plant. Faulconer was not trained for work at a chemical plant when employed, and received no training prior to commencement of work. His function was that of chemical operator in the surfactant plant. Faulconer was assigned one time to the monomer plant with a coworker. There was a spill while he was there from the reactor of about a quart of allyl alcohol. The almost immediate effect was to swell the eyes shut. The maintenance personnel washed down the spill accoutered in self-contained respiratory units. In the process, they opened the doors to vent the allyl alcohol vapors into the atmosphere. Faulconer, some ten feet away outside the building, could feel the effect of the allyl alcohol vapors on his sinuses and eyes.

The operations at the soap plant were plagued by continual breakdowns caused by lack of maintenance: overhead lines ruptured, pumps failed, and other malfunctions resulted. The toxic vapors released from the ruptures were simply vented into the atmosphere. When the sulphur burner in the soap plant broke down, so that sulphur dioxide fumes filled the building, the employees were instructed to wait until after dark, then open the doors and release the fumes into the atmosphere. At those times, the fumes became so thick that employees in the soap building had to wear respirators. The manufacture of the soap-based products in the surfactant plant involved sulphur and its compounds. Waste from that process included vapors of sulphuric acid, sulphur trioxide, and other chemical residues. It was a function of the acid scrubbers to cleanse the residues from the wastes before they were emitted, nontoxic and neutral, into the atmosphere through the stack. When the acid scrubbers malfunctioned, but production

continued nevertheless [as were the instructions of management], the vapors -still contaminated - and acid mist were emitted from the stack in the form of a plume, and sometimes as soap bubbles. The acid scrubbers malfunctioned 70% of the time. The smell from the plume was "very foul." When that residue descended, the grass turned brown, and irritated the skin it touched.

There were also repeated chemical spills. They occurred regularly, about once a week, usually at the start-up and shut-down phases of the production process. If the spill was in the plant, it was flushed down the drain and discharged into biopond one. The consequence was "a big pile of soap bubbles floating across the road" mixed with whatever toxic chemicals were in the pond. If the spill was outside the plant, as often occurred when trucks loaded or unloaded the chemicals, the spill was merely covered over with dirt. On one such occasion, some two to three thousand pounds of duodubonicsulfonic acid were released. Other outside spills resulted from the overfill of outside chemical storage tanks of multi-ton capacity. Another source of outside spills was from the storage of the liquid waste products. That residue [presumably because the liquid incinerator continued to malfunction] was placed in several thousands of drums outside the buildings for transport and disposal. Some of the drums leaked so that the contents filtered into Shaver Creek. Others would be smashed by the semi-trailers, and the contents laid open. Other drums exploded in the August sun and spewed the contents.

Faulconer described incidents when trucks came to be loaded to transport the surfactant products, but were refused by the employees because remnants of hazardous chemicals from the prior haul remained in the vans. The truckers simply drove a short distance off the premises, drained off the toxic matter, and returned for loads. It was a practice, Faulconer said, known by Alcolac and tolerated.

There was a logbook used to communicate between the production shifts. It contained instructions by production manager Gibbs as to the chemicals to be produced and the quantity. It was also used to report spills. Faulconer was instructed by Gibbs not to record a spill in the logbook until it was discussed with management first. He testified that only "ten, maybe twenty percent" of the spills he observed at the soap plant were recorded in the log. The spills usually recorded were the "non-hazardous type spills . . . five gallons of this, ten gallons of that." Those spills considered hazardous, Faulconer testified, were never noted in that log, or anywhere else, during his employment at Alcolac..

Faulconer confirmed the LeMaire testimony that Alcolac somehow got two or three days advance warning of Missouri Department of Natural Resources inspections, and that advisement was transmitted to the employees by production manager Gibbs. According to Falconer, the Alcolac management instructed Falconer and the others to "keep your mouth shut, don't volunteer any information." Falconer was discharged - he contended - two weeks after he contacted the president of the union at the Alcolac plant in Baltimore [the Sedalia plant was nonunion] for advice about better maintenance and safety procedures at Sedalia. He took that initiative, Falconer testified, only after he had approached Gibbs about better plant safety and was told "to keep [his] mouth shut."

C

Plant Operations After WAPORA

Alcolac dismissed WAPORA from the Sedalia plant project in December of 1979. The urgent WAPORA recommendations - for a

trained environmental engineer, employee training in pollution control, a restored liquid incinerator, the installation of eleven hydrocarbon systems to monitor emissions, and an aerator system sufficient to oxidate the increased influx of chemical waste into the bioponds - all remained unfulfilled.

Complaints by residents in the environs of Alcolac [some of them eventual plaintiffs, and others not] of odors, suds, fumes and bodily ailments from those and other emissions were made to Alcolac, to public officials, and then to the Missouri Department of Natural Resources [DNR] almost from the outset of operations. The DNR was drawn into contact with the Alcolac facility in Sedalia by a complaint of odors by Gwendolyn Lawrence, a nearby resident and eventual plaintiff. The response to that first complaint was by Ann Gessley, environmental specialist with the Division of Environmental Quality of the DNR in Jefferson City, Missouri. She arrived in Sedalia a week later to interview Ms. Lawrence - in August of 1978. The investigator on that occasion detected "a slight odor," and then proceeded to measure the odors around the circumference of the plant by means of a scentometer. That device allows a tester to measure the concentration of an odor by inhalations of comparative volumes of odor-free air [freshened by the carbon filter of the scentometer] and odorous [unfiltered] air. A dilution ratio of 7 to 1 between the filtered and ambient air constitutes an "excessive emission" according to the standards of the federal Clean Air Act enforced

by the DNR.[6] Ms. Gessley then entered Alcolac, informed then plant manager Fischer of the complaint, and was conducted through the facility. She had occasion to inspect the plant several times thereafter but acknowledged that she was qualified to enforce odor regulations, but not as to the "goings on of a chemical plant." Ms. Gessley testified that she never undertook to determine the source of the odors on the occasion of that first complaint, but her written report suggests that "the waste lagoon [biopond] could be the source of the odors." She doubted, however, that the biopond was the cause of the burning sensations the complaint of Ms. Lawrence described.

The recurrent complaints of odors and bodily discomforts from Alcolac emissions entailed frequent returns to Sedalia by Ms. Gessley and other DNR specialists. They conducted repeated surveillances around the Alcolac perimeter and often detected odors, but no violations - that is, odors diluted in a ratio of 7 to 1 by the scentometer test. Ms. Gressley acknowledged that the elapse of the two hours or so between the receipt of the complaint in Jefferson City and the arrival of DNR personnel in Sedalia "was why we couldn't get a violation." That is to say, whatever odors may have been emitted at the time of the complaint were often dissipated by the time an official test was possible. It was to allay that conundrum that the DNR empowered Sedalia officials - first the Director of Civilian Defense of Pettis County and then the Sedalia Police Department - to respond

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[6]The full evidence explains that when a scentometer inspection detected an odor in the dilution ratio of 7 to 1 or greater - measured at that concentration twice within the hour and fifteen minutes apart - an "excess emission" was established. A notice of "excess emission" was thereupon served upon the source [here, Alcolac]. The source was then allowed fifteen days to file a "start-up/shut-down or malfunction plea" - [apparently operational episodes which normally resulted in some period of excessive emission]. If the plea was rejected by the DNR, the "notice of excess emissions" was raised to the status of a formal "violation."

to complaints and to investigate by means of the scentometer. In a memorandum concerning a successive "walk-through" inspection of the Alcolac plant in October of 1978, Gessley did note "foaming problems" in Biopond No. 1, and noted also that the management "plans to reroute discharge [of] sulfactant plant to second and third cells [bioponds]." Gessley concluded her service with the DNR, and hence responsibility for the complaints of the Alcolac operation, at the close of 1979.

The response to the citizen complaints about the Alcolac operation, and the surveillances, by the DNR continued through Charles Crawford, a section chief of the air pollution control program, and others of his inspection group. Crawford testified that his division responded to over fifty complaints about Alcolac during his service in that function throughout 1979 and 1980. These responses then still originated from the Jefferson City office of the DNR, and Crawford participated in about fifteen of them. They comprised of surveillances and scentometer measurements. Crawford explained that, as to citizen complaints, because of the lag time between the receipt of the complaints at Sedalia and the arrival of the DNR in response, DNR "pretty much drew a blank on really getting anything to constitute a violation." He acknowledged on cross-examination, however, that in an official letter to The Sedalia Democrat, he commented that in the two years which preceded the abatement agreement between Alcolac and the DNR [on May 30, 1980] "we found several violations of the state odor regulation at Alcolac." There were also citizen complaints of foam as well as of physical ailments from the Alcolac operations.

Richard Nikkila, chief of the enforcement section of the DNR in 1979, and then in 1980 staff director for the air pollution control program of the agency, first made contact with Alcolac during a "walk-through" inspection of the plant with Ms.

Gessley. Such "walk-through" inspections were not to determine compliance, but simply "what is in operation and what [emission] controls are in place." In 1979 his office inaugurated the policy of increased surveillance of the plant for excessive odors, which Ms. Gessley and others then implemented. There were occasions when the scentometer measured excessive emissions, and Nikilla recalled that about four notices for such excesses issued to Alcolac from the DNR office from such surveillances. A series of notices of excessive emissions issued to Alcolac on citizens complaints as well.

On March 7, 1980, Nikkila served upon Alcolac a notice of excessive emissions. That investigation was prompted by the complaint of Ms. Lawrence in Sedalia to the DNR in Jefferson City of odors from the Alcolac plant. Nikkila and Hastings, another DNR official, arrived from Jefferson City an hour and a half later. The odors were palpable to them even without the use of a scentom-
eter, and both of them detected successive measurements at the 7 to 1 dilution ratio. They found the source: an exposed portion of the waste stream from the monomer building.

On March 14, 1980, an investigation of another complaint of odors from Alcolac resulted in another "notice of excess emissions." Alcolac was instructed to "determine the cause of the odor" and to achieve compliance by March 24, 1980. On April 30, 1980, Nikkila received two complaints of dark smoke and odors from the Alcolac surfactant plant. A nearby resident [and former plaintiff now deceased], Ms. Landon, complained that she had been overcome by odors some days before and became unconscious. Mr. Landon [also a plaintiff] complained that the emissions aggravated his emphysema. Nikkila came to Sedalia, took scentometer readings around the Alcolac perimeter and recorded measurements of 7 to 1 - "excess emissions."

On April 30, 1980, as a result of the investigation of a citizen complaint of dark smoke and odors from the Alcolac plant, Nikkila issued a notice of excess emissions. Nikkila gave notice of excess emissions to the laboratory technician, in the absence of plant manager Sutton. Alcolac interposed a "malfunction plea" against the April 30 notice of excess emissions which was rejected by the DNR after investigation. The official response by Nikkila for the DNR to plant manager Sutton for Alcolac on May 22, 1980 concluded.

> Since this is the third notice of excess emissions that you have received and the second which has been upgraded to a notice of violation, we intend to request issuance of an abatement order by the Director, Department of Natural Resources.

Sutton, in turn, issued a memorandum on that very date, May 22, 1980, to all Alcolac employees on the subject: "Odor and Pollution Control."

> During the past three months we have been cited three times by the Air Pollution Enforcement Division of the Department of Natural Resources for excessive odor emissions. These citations are serious and will probably result in further action from the DNR.[7]

The memorandum then gave the causes of the citations and complaints as "human error" in the operations which "could have been prevented had proper procedures been followed." It cautioned that "further operating errors or causes of odors will be very serious [and] could result in additional citations and ultimately the shut down of the plant."

These episodes prompted the DNR to issue an abatement order which directed Alcolac to cease the emission of odors in excess of the restriction imposed by its regulation. That order of abatement was superseded by an abatement agreement concluded

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[7] See APPENDIX G and H for the full text of the memorandum of the Alcolac plant manager Sutton to the employees.

between the DNR and Alcolac on May 30,1980. Its purpose [according to Nikkila] was to correct what the surveillances and investigations determined were the "significant sources of odor." Alcolac undertook, by its terms, to implement nine projects and procedures:

1. To install a single automatically timed hydrocarbon monitor to sample the emissions from each stack of the vapor incinerator and carbon adsorption system in the monomer building, equipped with a manual noise alarm when the limit for the hydrocarbon concentration is reached. The baseline data for the hydrocarbon concentration cut off limits was to be established by Alcolac and submitted to DNR for approval by December 1, 1980, and the alarm was to be installed thereafter by January 12, 1981.

2. To draft a written plan for the handling and control of the excess hydrocarbon concentration in the vapor incinerator and carbon adsorption system in the monomer building for DNR preliminary approval by November 10, 1980, and for final approval by December 1, 1980.

3. To implement by February 1, 1981 a system to determine on a one time basis the nature and concentrations of the emissions from the stacks of the vapor incinerator and carbon adsorption system for each product manufactured by Alcolac in the monomer building.

4. To install and operate an alkaline scrubber to remove 99.5% of the acrylates in the gaseous waste streams from the monomer building reactors and storage tanks. Alcolac undertook to report a completed and efficient operation of the installation to the DNR by June 30, 1981.

5. To install and operate an alkaline scrubber to remove 99.5% of the acrylates from the liquid waste streams from the hot wells in the vacuum system of the reactors in the monomer building. Alcolac undertook to report a completed and efficient operation of the installation to the DNR by July 15, 1981.

6. To develop and submit to the DNR by November 7, 1980 a plan and procedure for the routine analysis and changing of activated carbon in all carbon adsorption units so that the carbon is replaced before breakthrough of gaseous wastes.

7. To test, by November 24, 1980 and semi-annually thereafter, for emissions of sulfur dioxide and sulfur trioxide acid mist from the surfactant plant stack in accordance with the method approved by agency regulation.

8. To store - immediately - all drummed waste materials in either the monomer building, environmental control building or the warehouse.

9. To develop for DNR approval by November 15, 1980, a plan for the training of employees as to the control of odorous emissions from potential sources at the facility.

The abatement agreement did not prescribe any projects or procedures to further control emissions of odorous matter from the bioponds. Nikkila gave as reasons that the additional infusions of dilution water into the ponds and the installation of a system of aeration led the DNR to conclude that the bioponds "were no longer anaerobic, [s]o in our view, that was not an issue to take up with Alcolac."

It is the sense of the Nikkila testimony that the terms of the abatement agreement were met, with exceptions. Project 3, the system to determine on a one time basis the nature and concentrations of the emissions from the monomer building stacks, was never completed. Project 1, the hydrocarbon monitor, was installed but reported inoperative by Alcolac on December 8, 1981, and the manual reset alarm for that system was not actually installed until May 22, 1984. Project 8 called for the immediate storage of all drummed waste into one of the designated buildings which were designed to collect odors through the system of activated carbon. Alcolac, however, had adopted a plan to dispose of the physical inventory of 3000 drums at the rate of 500 per week, so that the removals would not be complete until July of 1980. A

number of those containers leaked ethyl acrylate and other toxic chemicals so that they were transferred to sound drums. Nikkila could not say how Project 8 was ultimately performed.

The abatement agreement notwithstanding, Nikkila acknowledged, the problem of odorous emissions was not resolved. The chemical spills continued also. The citizen complaints of odors from the Alcolac operation, the official detection of excessive emissions, and the citation for violations persisted. On August 15, 1980, a DNR investigation in response to a citizen complaint resulted in a notice of excess emissions to Alcolac. The Alcolac plea of malfunction was denied, and the emissions officially attributed to Alcolac negligence. In year 1981, the DNR files recorded notices of excessive emissions from a variety of operational incidents: foam off the bioponds carried beyond the Alcolac property; chemical spills, and from the malfunction of the carbon bed [and so officially excused]. The complaints of intrusive foam became so recurrent that then plant manager Sutton refused to accept any further notices of excess emissions from the DNR officials. Alcolac called Nikkila on May 14, 1981 to report a spill of allyl glycidil ether residue outside the monomer building to account for odors. An official inspection of the Alcolac plant in 1983 discovered off-premises odors in concentrations in excess of the 31 to 1 ratio. The complaints and official notices of excess emissions to Alcolac continued throughout 1983 and into 1985 -- a short time before the trial began.

In mid-1983, in order to improve the complaint response time, the DNR engaged the local government of Sedalia. Captain [then Sergeant] Rice of the police department performed that function until June of 1985. Rice was instructed in the use of the scentometer, made response to local complaints of odors, and maintained a log of those events. In the course of that two-year duty, Rice compiled 23 memoranda of complaint investigations made

by citizens in the environs of odors and resultant physical discomforts. These investigations, conducted night or day as the complaints were received, all led to the Alcolac operations. The odors were palpable to Rice, variously, as "similar to rotten eggs," "sewer odor," similar to "a hair permanent solution," and "foul odor of natural gas" - among other descriptions. The sources of these odors - except for a chemical spill - were the Alcolac bioponds, and most frequently, biopond one. The scentometer measured the odors on several occasions at the ratio of 31 to 1, and on one occasion, at the ratio of 171 to 1. On these occasions, the Alcolac supervisory personnel attributed the odors to a lack of fresh water in the ponds, the hurried release of soap byproducts, and inability to induce a breakdown of the residue from the production. Of the 23 complaints Rice investigated from June of 1983 to June of 1985, 14 were "excess emissions" as defined by regulation, and hence equivalent to violations unless excused.

Alcolac plant chemist Richardson explained that the natural system employed to degrade the compounds in the bioponds emits a "sulphur-type of odor." The odors are more recurrent in the early springtime when an "inversion occurs" - when the bacteria become more active and stir from the bottom of the pond to the top. It is a phenomenon which occurs about ten times a year, and sometimes the resultant foam is blown beyond the Alcolac premises. The chemist acknowledged that the ponds are not tested for toxic chemicals. [Nor, as Nikkila testified, was such a test ever undertaken by the DNR.]

Joseph Aid, a plant manager at Alcolac, acknowledged that the odors Captain Rice [investigator under DNR auspices] detected in June of 1983 in response to complaints were "[v]ery definitely from the bioponds." He attributed them, as well as the foam - as did chemist Richardson - to the soap component of

the wastewater and to the natural process of bio-oxidation employed to degrade that waste. He acknowledged at the trial [in late 1985]: "[T]here are still some odor and some foam problems, yes."

In July of 1980, some months after WAPORA was discharged from the Sedalia project in December of 1979, and within weeks of the execution of the abatement agreement on May 30, 1980, Alcolac employed Mark Blowers as environmental engineer -the first to occupy that position. Blowers, a recent graduate school environmental engineering laureate, was then 26 years of age and Alcolac was his first employment as environmental engineer in a chemical plant. He remained with Alcolac for two years - until June of 1982. His resonsibility centered around the environmental control building. Blowers replaced an environmental control technician, McCutcheon - a college graduate, but without other qualification in environmental control matters. His duty was also "to keep an eye on the environmental control building and make sure everything was going okay down there." Blowers acknowledged that Joseph LeMaire, the maintenance worker, had served as environmental control technician, but then was returned to maintenance work.

It befell Blowers to serve as plant liaison with governmental agencies, such as tne DNR and the EPA, and hence to implement the abatement agreement concluded between Alcolac and the DNR just the month before. To that end, Blowers undertook to establish procedures for spill control and clean-up. He also undertook to establish procedures for the treatment and storage of hazardous waste so as to comply with the provisions of the federal Resource Conservation Recovery Act [RCRA], then coming into effect. Blowers was also the official designated to respond to citizen complaints of Alcolac emissions. In those roles, Blowers was in frequent communication with Nikkila and other

officials of the DNR. It had been the procedure to maintain the record of spills in the daily reports of the foreman - entries interspersed with other notations on plant operations. Blowers collated these spill notations from the daily reports into a log "strictly for spills" and maintained that system from December of 1980 through December of 1981. Thereafter - for no explained reason - the log was discontinued.

The log reported 111 spills during that year of December, 1980 to December, 1981. The quantity of the spills, as recorded in the log, ranged from one quart to 2900 gallons of the particular compound. Blowers surmised that during the two years he served with Alcolac at Sedalia, "an environmental concern-type spill, major spill, would [occur] once a month." The small chemical spills in the environmental control building were rinsed down the drain into the sump pit, and small spills in the monomer building were treated with 3M absorbent cloth. That special cloth functioned to absorb the organic matter from the surface of the waste, and the cloth was then disposed of in drums and discarded as solid waste. Blowers acknowledged that there were occasions when the employees neglected these clean-up procedures, and hence the carbon filter systems were bypassed, so that the spills went directly into biopond one - still untreated and toxic. On other occasions the sumps in the environmental control building would simply overflow from more production of waste in the monomer building than the pumps could accommodate. In such event, the environmental control building operated "in a bypass mode" and diverted the chemical waste into the biopond without filtration. Leaky drums were also a source of chemical spills. These fifty-five gallon metal drums, used to store toxic wastes and stacked on the grounds, were sometimes eaten through so that the liquid exuded. They were also struck by trucks and severely damaged so as also to become "leakers."

Another objective of the abatement agreement Blowers was engaged to implement was that Alcolac install a system of alkaline scrubbers to remove the acrylates from the liquid as well as gaseous waste streams from the monomer reactors. The purpose of the scrubbers was to expunge those toxic residues, as well as their odors, from vapors and liquids before they were vented to the carbon bed and fume incinerator. Alcolac had operated without such scrubbers for the two years which anteceded the abatement agreement. They were installed during his tenure as environmental control engineer, and once in operation - as Blowers described it - they "worked fairly well most of the time."[8]

Another objective of the abatement agreement - the installation of the hydrocarbon monitor on the fume incinerator and carbon bed stacks of the monomer building - was also undertaken during his tenure at Alcolac. The monitor broke down occasionally, but even when the device operated [Blowers said], there was a "major problem [in] trying to determine a meaning to the data [derived] from the hydrocarbon analyzer." The monitor measured carbon atoms, rather than specific molecules. Thus, although the monitor could identify the carbon content of an organic emission, it could not identify the particular compound emitted. For instance: the detection of four carbon atoms in the emission could signify the presence of epichlorohydrin or any other compound of similar molecular structure, but could not identify which one. It was a delineated objective [¶3] of the

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8 The surfactant factory was also equipped with scrubbers - acid scrubbers - designed to remove chemicals from the vapor wastes before they were emitted through the surfactant stack. There was evidence that the acid scrubbers functioned only 30% of the time, with the consequence that odors and substances emitted into the atmosphere. The acid scrubbers were not a subject of the abatement agreement.

abatement agreement of May 30, 1980, that Alcolac install by February 1, 1981, a system to determine the "nature and concentration of the emissions from the stacks of the vapor incinerator and carbon adsorption system for each product manufactured by Alcolac in the monomer building." That system was never installed.

Blowers also dealt with the complaints of citizens and of officials concerning the Alcolac operations. The recurrent complaints were of odors and foam from the bioponds. He estimated two dozen such reports during the two years with Alcolac. He explained that the emission of the soap material into the bioponds was an incident of the normal operation of the surfactant plant. The foam - as other Alcolac witnesses also undertook to explain - was the result of the aerator action, and so was not an unexpected occurrence. The foam on occasion built up to a height of 15 to 20 feet on the pond, and when the wind rose, "bl[ew] all about." He recalled that on about two occasions, he saw the foam on the property of Alcolac neighbors. The foam came into contact with Blowers and other employees at times, but never with deleterious effect - Blowers testified. He acknowledged, however, that during his tenure the bioponds were never tested for toxicity, so he could not say whether they were contaminated.

In addition to the continued malfunction of certain pollution control equipment [such as the liquid incinerator at the monomer plant and the acid scrubber at the surfactant plant], the recurrence of chemical spills, and complaints of odors and foam from the bioponds, Blowers encountered two episodes of rupture of the reactor in the monomer plant during his two-year tenure at Alcolac. They involved the blow-out of the rupture disk - a sort of safety release to relieve against excess pressure generated in the reactor tanks during the chemical production process. The rupture disk is a cartridge insert between the

reactor and pipeline vent. It is the theory of design that when the pressure in the reactor becomes excessive, the cartridge ruptures and releases the excess gas into the atmosphere through the pipeline. In the early morning of September 25, 1981, a rupture disk blew out, but instead of gas vented into the atmosphere through the pipe, the explosion [as Blowers reported the incident to the DNR], "took the pipe with it and also damaged [the] building." The report identified the chemical component in the reactor at the time of the incident as diallyl maleate and described that compound as "a mild eye irritant ... toxic only through ingestion." Blowers continued to reassure the DNR that there was "no reason to expect toxic effect" from the spewed debris. The city officials evacuated the neighbors in the path of the wind direction and the Sedalia Fire Department was called to the site.

The investigation established that the rupture disk blowout not only "took pipe with it" - as described by the Alcolac report - but also a portion of the monomer plant roof as well. Nor was the debris from the explosion confined to the Alcolac premises, but rather was strewn to the north and south onto private property. A number of the firefighters who responded to the call reported skin irritation, and another fell sick. The diallyl maleate in production at the time of the explosion, Alcolac disclosed, was being made from allyl alcohol and maleic anhydride. Blowers acknowledged that allyl alcohol was toxic and could cause severe irritation to the body and if absorbed through the skin, could cause injury or death. The symptoms displayed by the firemen, the DNR investigation established, "matched the toxicological information given by the [treating] doctor."

At the time of the explosion of September 25, 1981 Alcolac was without a plant manager. Sutton was gone and Aid, his successor, had not yet arrived. Aid came to Alcolac on October 19, 1981 - a month after the explosion. This suit against Alcolac had been commenced by then and pended. Among his first initiatives, Aid addressed a memorandum to the supervisors and lead operators of the Sedalia operation concerning the reporting of spills:

> I want to emphasize the importance of reporting all spills at the time they occur. Our liquid waste treatment system only has limited flexibility, and slugs of material create serious problems. . . . If a spill occurs, you must be made aware of it by your operators and a report must be made.

Notwithstanding the hortation of that official memorandum, it was the testimony of lead operator Buckner that spills occurred in the monomer building virtually every day, many of them unreported. The workers found it more convenient to "wash it down than fill out the paperwork." The clean-up procedures were also discouraged as too costly. The prescribed method for the disposal of most spills was absorption by organic [3M] cloth - but "supervisors complain[ed] it was too expensive and too costly and it was just kind of pushed over." It became the practice, rather, to wash the spills into the drain to the environmental control building. That occasionally caused the environmental control building sumps to overflow with the consequence that the spills liquid was coursed around the filtration system and, still toxic, was pumped directly into biopond one.

Buckner came to Alcolac in September of 1980 and remained there for three years - until September of 1983. Thus, much of his tenure coincided with that of environmental control engineer Blowers. Buckner worked exclusively in the monomer building, where the toxic chemicals were used in production. They

included epichlorohydrin, allyl alcohol, methylene chloride, dimethyl sulfate and methyl chloride. He was given no formal training in the use of chemicals, but learned as he went. [Epichlorohydrin, he learned, had no odor and is invisible. "It is just there and it burns you."] Buckner - as did Blowers - confirmed that the foam in biopond one often reached a height of fifteen feet. Buckner - in contradiction of Blowers - related ten to twelve as the number of rupture disk blowout incidents in the monomer building during his employment there. It was also the practice, Buckner testified, to complete the run of a "batch" of chemicals before the break of daylight so that the toxic vapors generated from that production process could be vented during the dark "so nobody could see it."

There was the testimony of another chemical operator at the monomer plant - called as a witness by Alcolac - Charles Henderson, who had been employed continuously since production began at Sedalia. He testified to occasional spills in the monomer building - and described a "spill" as "something that would take manpower and maybe, you know, two or three hours to rectify." Anything less, he considered a "leak." A leak occurred two or three times a week, and was absorbed by 3M paper or simply washed into the drain. A spill occurred very infrequently - two or three times during his eight-year tenure. They occurred outside the building, and were promptly washed down into the drain connected to the environmental control building. Henderson could not estimate, however, the number of toxic spills of lesser magnitude than those which required several persons to "get it cleaned up rapidly." The witness considered the 111 spills reported in the 1981 log maintained by Blowers sounded "awfully high but ... possible." He acknowledged that during those eight years of employment at Alcolac, he had never been shown the environmental control systems operating manual prepared for the Sedalia plant by WAPORA. He had

responded to counsel that he suffered from no Alcolac-related health problems, but acknowledged a recent medical examination report which disclosed complaints of numbness or tingling of the hands and feet. The witness conceded an interest in the outcome of the suit in which he gave testimony: "It means my job if they [Alcolac] lose."

There was other testimony - through Alcolac witnesses -as to the operation and maintenance of the environmental controls at the Sedalia plant. That evidence was interstitial of the proofs already described and undertook to confirm the defense theme that the internal environmental control procedures were methodically observed and were effective to meet official standards. The responses to cross-examination raised some inferences of contradiction. This spate of testimony, for the most part, related to fragments of the span during which Alcolac operated.

Richardson was plant chemist at the Sedalia Alcolac for five years [from 1980]. He had worked for the Alcolac corporation for twenty years altogether, and suffered no significant health problem as a result of that employment. He worked with monomers in the laboratory, in safety garb. It was his testimony that the odors perceptible in the plant - "sulphur-type" - were not from the chemicals, but from the bioponds, and were the natural incident of the biodegradation process. The foam on the ponds was a product of the soap plant, and [presumably] not of toxic chemicals. Cross-examination elicited that neither Richardson as plant chemist nor any other employee ever sampled the ponds for toxic chemicals during his stint with Alcolac. He acknowledged that laboratory tests in 1984 disclosed that he had developed a physical condition of abnormal globulins, and that in 1985 abnormal hemoglobin was detected. Richardson conceded "an interest in the outcome of this lawsuit to the extent that [he] would like to see Alcolac win the case."

Lloyd Goode was the environmental control operator during virtually the entire span of the Alcolac operation in Sedalia. His employment progressed from maintenance, to the surfactant plant, to environmental control building as operator. This progression of duty was without any training. It was his function to run tests and otherwise "take care of the bio-oxidation ponds." He testified that there were fish in the ponds and other animal life frequented them. He acknowledged on cross-examination that the tests he conducted for the ponds did not include any for toxic chemicals. He, too, admitted an interest that Alcolac win the litigation.

James Wells, a DNR supervisor, performed a hazardous waste management compliance and complaint inspection. Wells testified to an inspection of the Missouri Pacific Railroad installation located somewhat to the south and west of Alcolac. The inspection, conducted on May 31, 1985, was to determine compliance with federal and state hazardous waste management laws and regulations. Missouri Pacific was registered with the federal EPA as a hazardous waste generator, but not with the state DNR. The inspection identified several potentially hazardous wastes and resulted in a notice of violation for the generation and disposal of such wastes without registration with the state DNR. Wells acknowledged that the DNR had received no complaint of any Missouri Pacific activity, and further, that the tests conducted on those premises had not yet been reported. Wells also had occasion to be on the Alcolac premises - both for routine hazardous waste management inspections to determine compliance with the newly enacted federal Resource Conservation and Recovery Act as well as in response to citizen complaints. The inspections for RCRA compliance were confined to the management and storage of toxic wastes, and did not relate to the condition of the bioponds or emissions from the production process. Wells conducted three such

inspections at Alcolac, between June of 1981 and June of 1985. Each of the inspections noted some unsatisfactory feature - among them, the lack of documentation that "personnel training [was] conducted annually" and the storage of corrosive waste in damaged containers. The inspections, nevertheless, found Alcolac in substantial compliance with the RCRA. Wells also dealt with air pollution control at the DNR and responded to six citizens complaints about air pollution - odors and foam - from the Alcolac operations.

Raymond was another DNR official - the chief of technical support of the air pollution control program. It was the function of the technical support group to collect information and surveys from Missouri plants which generate air contaminants and, on the basis of the information furnished by the company, to calculate an "emission inventory" for that particular plant.

On the basis of the information furnished by the respective companies, DNR calculated that for year 1983 Alcolac generated two tons of emissions of total hydrocarbons compared with twenty-nine tons of emissions of such compounds generated by the Missouri Pacific Railroad yards nearby. The saliency of that evidence, as the inquiry itself suggests, was to demonstrate the "efficiency of [the Alcolac] control devices." The function of the inventory, as the witness repeated with emphasis, was not to "evaluate companies," but to develop a "monitoring network [in] major areas of high concentration of pollutants." That national data base, under the aegis of the federal Environmental Protection Agency, depends altogether upon what information the particular company chooses to disclose. The concern of the DNR, in any event, related to emissions of volatile organic compounds, and not to total hydrocarbons, as such. The production processes at Alcolac, the DNR

witness acknowledged, generated volatile organic compound emissions. The "emission inventory" of volatile organic compounds for year 1983 did not list Alcolac, the witness agreed, because "Alcolac did not choose to send [DNR] any written information about any volatile organic compounds."

Hughie Clay came to Sedalia in April of 1985 as plant maintenance engineer at Alcolac. Suit then pended and at the time Clay testified, he had worked at Alcolac for seven months. His work function was to maintain the plant installation and attend to upkeep and repairs. Clay performed no significant duty. Blowers had since left Alcolac, and the plant operated without an environmental control engineer during the period Clay was employed there. The environmental responsibility, rather, reposed in plant manager Aid. The description given by Clay of the production and environmental control processes during his brief span at Alcolac depicted a plant of sophisticated design and virtually flawless operation. Cross-examination elicited, however, the incidence of two fires in the monomer building during his seven-month span of employment. He acknowledged also that he was not informed about past systems malfunction at the plant, and that even the most perfectly designed safety feature could be rendered irrelevant if bypassed.

D.

1.

Opinion for the Plaintiffs

The Alcolac Operation: Opinions of the Experts

The plaintiffs presented Frederick W. Boelter, industrial engineer and staff expert on industrial hygiene with Carnow-Connibear Associates,[9] for his opinion on the safeness of

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[9]Carnow-Connibear Associates is a medically based consultation firm which provides services to governments as well as private clients on a broad range of occupational and environmental health subjects. The staff associates are experts in numerous disci-

the Alcolac operations to the residents of the environs. His opinion rested on numerous evidentiary sources, among them: the WAPORA document; schematic designs of the facility; observations of the physical Alcolac facility; air and water tests conducted on and near the plant premises; analysis of the plant production documents for the past seven years [1978 to 1985]; analysis of the raw materials, production processes and intermediate chemical products; official documents and memoranda between the Missouri Department of Natural Resources; and the internal records of Alcolac. To derive opinion, the witness interrelated the evidence from these sources to the environmental control practices described by the several Alcolac employee witnesses.

Boelter gave opinion that, despite the state of the art design of the environmental controls installed at Alcolac, the neglect of these procedures by the employees as well as the malfunction of the control equipment, has resulted from 1978 in an operation that has not been safe to residents in the environs from exposure to toxic chemicals and compounds.

Boelter elaborated: the lack of trained personnel, the practice of the Alcolac employees to use the bypass equipment to route the monomer toxic liquid waste stream around the filter system so as to discharge directly into biopond one, the practice of the employees to discharge the surfactant liquid waste stream into biopond one [instead of biopond two as the design intended] so that the toxic waste intermixed with the soap waste with the resultant risk that the toxic compound would be borne into the atmosphere, the failure to control emissions inside the buildings and allow the vapors to vent into the atmosphere through open

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plines - among them, toxicology, epidemiology and industrial hygiene. The witness Boelter served as director of environmental services for the Associates.

doors, the want of a valid spill control program, the falsification of records, the lack of a standard operating procedure - all bespoke a management "terribly lax in addressing basic plant operation requirements" and were contrary to industry standards.

Boelter also related his opinion of unsafe operation to the log of chemical spills kept for the year 1981 by Blowers during his service at Alcolac as environmental control engineer. The log recorded 111 spills - among them quantities of such extremely toxic chemicals as epichloroydrin, allyl alcohol and allyl methacrylate. The log explained the cause for each recorded spill, and a number of them - as interpreted by Boelter -were the result of a want of standard operating procedure, employee neglect and equipment leaks.

Boelter also determined from the history of equipment malfunction and misuse, that Alcolac was from inception in 1978 an operation unsafe to nearby residents. The incinerator designed as the environmental control for the disposal of the hazardous liquid waste from the monomer process never functioned properly, and was abandoned in 1981. In that interim, the malfunction frequently resulted in incomplete combustion of the waste and hence emission of that, and other, toxic residue into the atmosphere. The fume incinerator used for the combustion of the vapor wastes in the monomer building also did not always function so that toxic wastes were emitted from that source also. The acid scrubbers in the surfactant building often did not function with the consequence that the bioponds were further stressed by the additional soap and their normal biogradation function impaired. When the acid scrubber malfunctioned, also, sulphur dioxide, sulphur trioxide and sodium hydroxide - all regulated by law as pollutants - were emitted in the form of a plume of dark smoke.

Boelter also rested opinion on the recurrent overload of the bioponds with much higher quantities of organic wastes from the monomer building than the design contemplated [noted by WAPORA as early as 1978], with the consequence that the biodegradation of the chemical wastewater was impaired. These chemicals as well as the surfactant deposits from the faulty acid scrubbers, the evidentiary sources disclosed, produced odors which were never altogether dispelled by the installation of aerators in the bioponds and the use of ammonium nitrate. These sources of evidence also disclosed to Boelter that the bioponds were never monitored for toxicity either of the liquids or vapors.

Boelter rested his opinion that Alcolac was an operation not safe to the nearby residents on yet another ground: the lack of detectors on the stacks and carbon beds to monitor on a "product-to-product" basis to identify the precise hydrocarbon compound - and its toxicity - being emitted from the production of the particular monomer batch. It was a recommendation made early by WAPORA, but rejected by Alcolac as too costly. It was later a subject of the abatement agreement with the DNR in 1980, but never implemented by Alcolac. The documents Boelter reviewed disclosed also that the carbon adsorption systems, both in the monomer building for the vapor wastes and in the environmental control building for the liquid waste, were allowed to become oversaturated so that the excess wastes simply passed through "as though there were no device in place at all."

The opinion also rested on three tests conducted by Boelter and the Carnow-Connibear Associates on and around the Alcolac premises.

The first was conducted on September 12 through September 14, 1983 at residences of several plaintiffs around the plant. Air samples were taken at those sites for lead, sulfates,

methanol and other organic compounds. They determined that Alcolac contributed to the level of sulfates, but were inconclusive as to methanol and the other compounds. Boelter explained that the testers had no real knowledge as to what was in the process of manufacture on those days, therefore the sampling was "essentially blind."

The second survey was conducted on September 4 through September 7, 1984, and involved both air and water tests. The water at the residences of six of the plaintiffs was tested, as was the ambient air, to determine any detectable level of six specific toxic compounds. The tests of the samples disclosed no significant concentration of any of those six contaminants. Boelter explained that on the days of the tests, the plant was "not operating in a way to determine the level of activity and again, the compounds that were being used and what was being manufactured at the time was unknown to us."

The third survey was conducted on June 22, 1985, on the Alcolac premises under the auspices of a court order. There was no plant activity on that day, and hence air samples, Boelter explained, would have been of no use. [The Alcolac records for that day and the several days before indicate that the reactors were in the process of restoration and otherwise inactive.] Water samples were taken from bioponds one and two. Toluene, a toxic compound, was found in a concentration of 240 parts per billion, as well as other compounds, one known as two-three dichloropropene and another called three-three oxybispropene. The significance of the presence of toluene [although in relatively low concentration], Boelter explained, is that that toxic compound is very volatile and so indicates a more massive recent presence. The significance of two-three dichloropropene is as a "footprint" for epichlorohydrin, a very toxic compound. The chloropropenes,

Boelter explained, are a degradation product of epichlorohydrin and indicate that epichlorohydrin was present. Boelter explained further that it is impossible to sample directly for epichlorohydrin in water since it is so volatile. "It has a very short half life in water" and disappears very quickly either by evaporation into the air or by decomposition into other products such as dichloropropene. Propene [also known as allyl ether], a toxic organic compound also found n the bioponds water samples, is formed from the reaction between allyl alcohol and sulfuric acid - chemicals used in the Alcolac production process. The tests detected another toxic compound in the biopond waters - a nitrosamine called N-nitrosomorphyoline, the result of the heavy use of ammonium nitrate in the bioponds to dissipate the odors. The detection of the presence of the morpholine compounds is significant, Boelter explained, because they are associated with the manufacture of cosmetic chemicals - the production of the surfactant plant.

Boelter concluded that the presence of these organic compounds in the bioponds was "of particular concern" because the samples were taken under conditions much more favorable than "historical conditions,"[10] and if what was found was any indication "the conditions historically were more than likely much worse than this and with the difficulty in controlling the ponds and the foam that is generated, this could be a very significant contribution to the effects on the surrounding environment." Boelter observed that only in the chemical and industrial plants of "third world countries" had he seen "this bad of a situation in one place."

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[10]The entry onto the Alcolac premises for testing purposes under the court order was deferred for a week. On the day of the tests, and for some days before the Alcolac records show, the monomer reactors were reconditioned and there was no production activity.

## Opinion for Alcolac

Alcolac presented the deposition testimony of Robert B. Jacko, Ph.D., expert in environmental engineering and faculty member of the Purdue School of Civil Engineering. The witness also had conducted research in the flow of toxic pollutants in chemical and other facilities, but that inquiry did not include health studies.

Dr. Jacko made three visits to the Alcolac facility. The first two were in the autumn of 1984, and the third was on June 22, 1985, the same date the Carnow-Connibear Associates conducted tests on the Alcolac premises. In preparation for these visits, Jacko was furnished with numerous documents and other information - which included the environmental study conducted by the Carnow-Connibear Associates off-premises in the vicinity of the Alcolac plant. Jacko was also furnished the specifications of "all the environmental control systems at Alcolac, as modified by Alcolac employees to update them." It was on the basis of that information as well as his observations and over-flights of the facility that the Jacko testimony and opinions are based.

It was the opinion of expert Jacko that Alcolac functioned as a closed system for the containment of volatile and organic vapors. That is to say, the environmental control devices operated so that the vapors were not released into the atmosphere until the organic matter had been expunged. In the warehouse building, in case of a chemical spill, the building was completely sealed and the fans carried the vapors to the carbon beds for adsorption. In the monomer building, a system of ducts captured the vapors and conducted them to the carbon beds for adsorption, and the vapors are then emitted through the fume incinerator. In the surfactant building, one acid scrubber removes the sulphur

dioxide and sulfur trioxide before the vapors are emitted through the stacks, and the other, the solid particulate matter. The emissions from this operation, the witness testified, conformed to the EPA standards. In the environmental control building, the vapors from the monomer liquid collected in the sumps ran through a carbon adsorption system, then through the caustic scrubbers,[11] into the fume incinerator, and then emitted into the atmosphere.

Jacko conducted no tests, either of the ambient air or of the bioponds. He "walked the ponds" with Boelter on the June 22, 1985 occasion, but "perceive[d] no significant odors at that time." Jacko mentioned two monitors on the bioponds, one to measure biological oxygen demand, and the other to measure dissolved oxygen. He noted no Alcolac procedure or practice to test the bioponds for toxicity. The witness alluded to a sulfur emission test conducted on June 20, 1984 by the Shell Engineering Associates on behalf of Alcolac. The test was for both sulfates and sulfur dioxide, as required by the abatement agreement with the DNR, and certified that the emission rates of both samples were in concentrations lower than the maximum allowed limits. The witness also alluded to the extensive forms submitted by Alcolac for air pollution permits then issued by official agency, presumably as evidence of valid environmental practices.

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[11]The environmental control systems Jacko observed and described were those as "updated" and in place at the time of his observations and environmental appraisals in 1984 and 1985. The caustic scrubbers were installed in the monomer building as a result of the May 30, 1980 abatement agreement with the DNR. The liquid incinerator [according to Blowers], never effective as the environmental control device for the disposal of hazardous waste, was no longer in use after mid-1981. Accordingly, the testimony of Dr. Jacko does not allude to that mechanism, nor does his opinion that Alcolac functioned as a "closed system" consider the evidence of the malfunction of the liquid incinerator from 1978 until its use was discontinued, nor the concerns expressed by WAPORA and the DNR to Alcolac of the adverse environmental effects of the continued malfunction of that device.

Jacko mentioned no adverse environmental consequence from the past acknowledged malfunctions of the environmental control system as "updated" - as of the acid scrubbers in the surfactant building and the hydrocarbon monitor installed under the abatement agreement. It is evident that the deposition opinion Jacko rendered - that Alcolac functioned as a closed environmental system as to volatile or organic vapors - was essentially an appraisal of conditions observed in 1984 and 1985.

Alcolac also presented the testimony of Harvey Shell, chemical engineer and president of Shell Engineering and Associates. Shell Engineering conducted a test of the surfactant stacks at Alcolac on June 20, 1984, and of the monomer fume incinerator stack on June 26, 1984. The surfactant test was undertaken to comply with the term of the abatement agreement that Alcolac would test the surfactant stack semi-annually for emissions of sulfur dioxide and sulfur trioxide acid mist. The surfactant tests certified that the emission rates were in concentrations less than the maximum allowed by regulation. Shell acknowledged that the test was for total hydrocarbons, and not for emissions of specific organic compounds. Hence, what the emissions were, or if they were toxic, were not determinable by the test.

Shell took no test of the biopond for toxicity, nor of the foam. Nevertheless, he disagreed with the conclusions of the Carnow-Connibear test that the quantity of toluene measured in the biopond was a significant source of toxic emission. He concluded, rather, that "the ponds are working and doing what they are supposed to do."

He rendered the opinion - based upon reports submitted by Missouri Pacific to the DNR that it generates twenty-nine tons of emissions per year in total hydrocarbons and in volatile organic compounds - that the Missouri Pacific operation contributed

to the ambient emissions in the area of Alcolac. Shell acknowledged he had undertaken no tests of Missouri Pacific emissions.

## III

## The Chemicals

Alcolac produced surfactants and monomers. The surfactant production, for the most part soaps, was a continuous operation. The chemical ingredients as well as the wastes, accordingly, were constant.[12] The monomer production, on the other hand, was custom designed to the needs of the customer, and production depended upon demand. The chemical ingredients of the monomers, therefore, varied according to the order. Many of these chemicals were toxic - as were their wastes. Of these, some were rated as hazardous by the Environmental Protection Agency.[13] Others of the many chemicals used in the monomer production may have been more toxic and hazardous than those identified in the testimony, but have never been tested for toxicity, so that the effects of these

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12 The constant wastes from the surfactant process were sulphur dioxide, sulphur trioxide, an acid mist of sulphuric acid and some sodium hydroxide.

13 TOXICITY AND HAZARD: "Toxicity is defined as the ability of a chemical to cause injury once it reached a susceptible site in or on the body. Hazard is defined as the likelihood that a chemical will cause injury under circumstances of ordinary use." N. Sax, Dangerous Properties of Industrial Materials 2 (6th ed. 1984).

agents on the biological system are not known.[14]

Among the chemicals regularly used by Alcolac in the monomer production process, and already tested for toxic effect, were epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, glycidyl ether and cyclohexene. Of these six chemicals [Dr. Legator testified[15]], five are designated as hazardous by the EPA, and three are known carcinogens. The effect of a toxic chemical on the human body, Dr. Legator explained, depends upon the concentration of the chemical and duration of the exposure to it. Or, as rendered into a formula: "Concentration times time equals effect." That equation is subject to variables - among them, the genetic susceptibility of the person to chemicals and the mode of life adopted by the person. The exposure may be chronic - over a prolonged time, or acute - over a short time. The toxic effects may also manifest as chronic or acute. There are diverse portals through which the toxic chemicals gain access to the body. The major routes are through the skin, the mouth or lungs. A toxic chemical may characteristically attack a particular organ or may target multiple organs. When absorbed, toxic chemicals can affect the immune system and, if the dose is high and prolonged enough, can cause mutations in the human body.

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The toxicology of the Alcolac chemicals was the subject of the testimony of expert witnesses Dr. Marvin Legator and Dr. Betram Carnow for the plaintiffs, and Dr. Sorrell Schwartz and Dr. Edward Emmett for the defendant Alcolac. That testimony established that the Environmental Protection Agency [EPA] toxic substance list numbers 130,000 industrial chemicals as toxic. Of that number less than 5% have been studied for toxic effects, and less than 1% have been studied for carcinogenic or mutagenic effect upon humans.

15

Dr. Marvin Legator, Director of Division of Environmental Toxicology at the University of Texas Medical Branch and erstwhile professor of genetics - among an array of professional credits - was presented by the plaintiffs for expert testimony on environmental and genetic toxicology.

Where the exposures are from multiple toxicants [as in the monomer production process], Dr. Legator explained, the combined toxic effects from the interaction may be much greater than the mathematical sum of their individual effects. As toxicologist Schwartz[16] commented, science recognizes chemical interactions where "one and one equal ten." That effect is known by the technical term synergism. Another means whereby toxic chemicals may become more toxic is by the process of pyrolysis. That term means merely breakdown by heat. Incineration was the form of pyrolysis used by Alcolac to dispose of some toxic wastes. If the incineration is at a heat effective to destroy the chemicals [Dr. Legator explained], the toxic effects are also destroyed. If the incineration is incomplete so that the chemicals are only partially destroyed, the pyrolysis may result in more toxic chemicals than originally and "produce more chemical in the atmosphere." Dr. Legator gave as opinion - in terms of pyrolysis - that the effect of the operaton by Alcolac of the liquid incinerator at low heat levels because of continued malfunction was the discharge of more toxic substances.[17]

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Dr. Sorrell L. Schwartz, Professor of Pharmacology at Georgetown University Medical Center, Scientific Director for the Center for Environmental Health and Human Toxicology at George Washington University - among an array of professional credits - was presented by Alcolac for expert testimony on toxicology and pharmacology.

17

It was an opinion confirmed by Jacob Bregman, president of WAPORA, and architect of the environmental control systems installed at the Alcolac plant:

> Pyrolysis, the term means breakdown by heat, and when you take a chemical and put it in an incinerator, or heat it up in any way, the chemical breaks down. That's called pyrolysis.
>
> Depending on the chemical, the eventual breakdown products are carbon dioxide and water. However, it goes through a whole series of smaller chemicals,

Dr. Legator then discussed the toxicological role of each of the six chemicals used in the monomer production process at Alcolac and already tested for toxic effect:

EPICHLOROHYDRIN

Alcolac used some 378,000 pounds of epichlorohydrin per year. It is listed by the EPA as a direct carcinogen, a hazardous substance, and a hazardous waste. It is a volatile chemical and enters the human system through inhalation and also the skin. The target organs are the brain and the central nervous system, as well as the kidney and liver, among others. It affects the blood and causes porphyria - a disease of liver metabolism. The chemical is also a mutagen and induces "transmissible genetic damage." It has also been identified as the cause, even at "very low concentrations," of chromosome damage in humans - that is, to "[t]he package that holds our DNA." That is significant, Dr. Legator explained, because "literally every chemical that I know of that causes chromosomal damage also causes cancer." It is an identification of cause, moreover, borne out by epidemiological studies. Animal studies confirm that epichlorohydrin causes a variety of cancers in different organ sites, breaks down the chromosomes, and affects the reproductive system.

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> some of which may be toxic, some of which may not be toxic, and they all eventually break down. Those are pyrolysis products.
>
> If the incinerator isn't operated properly, not only do some of the original chemicals get out, but some of these breakdown [sic] products.

Dr. Bertram Carnow, another expert in toxicology and other sciences,[18] described epichlorohydrin as a direct carcinogen - "an extraordinarily active chemical," a "grabber" which "grabs onto DNA" and changes it. The chemical also deforms the immune system so that it cannot respond to the attacks on the DNA by cancer cells. Dr. Carnow confirmed that the indication of an earlier epidemiological study that exposure to epichlorohydrin presents a risk of cancer to humans has been "more strongly proven." The witness considered it significant that Alcolac has used an average of 378,000 pounds of the chemical in each of the past seven years, since even "[i]n very small quantities, it is extraordinarily toxic." The volatility of the chemical, moreover, is such that if "put out into water, [i]t will go from water into air." A test of Alcolac biopond one by the Carnow professional group found "the footprints of epichlorohydrin" in the waters.

The toxicologist for Alcolac, Dr. Sorrell Schwartz, agreed that epichlorohydrin can cause damage to multiple organ systems - the liver, the lung, the kidney and other sites. He would not acknowledge it as a "foregone conclusion," however, that the compound can cause porphyria, but considered it "certainly plausible," since the chemical "does have a fairly extensive ability to interact with the systems, with the liver." The witness agreed that epichlorohydrin, as an alkylating agent, is a "cell-killer," but maintained that it has not been demonstrated that the chemical depresses the immune system in humans. Dr. Schwartz agreed that chronic effects may result from exposure to

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[18] Dr. Bertram Carnow, a partner in the professional consultation firm, Carnow, Conibear & Associates, was the most prominent expert witness presented by the plaintiffs. His testimony ranges over some ten volumes of transcript. A fuller and more considered discussion of the expert qualifications and testimony of Dr. Carnow is rendered in the course of opinion.

epichlorohydrin in adequate doses and that the chemical is a proven animal carcinogen. He concluded: "It is possible that it can cause it in humans." Indeed, it was the opinion of Dr. Arthur C. Zahalsky, presented as an expert on immunology by the plaintiffs, that epichlorohydrin is not only a carcinogen, but acts adversely on the human immune system.

The opinions of Dr. Edward Anthony Emmett, another expert witness for Alcolac,[19] were somewhat at variance with those given by Alcolac toxicologist, Dr. Schwartz, as to the toxic effects of epichlorohydrin. Dr. Emmett acknowledged that recent publications conclude that exposure to epichlorohydrin presents a risk of cancer to humans. He agreed with the other witnesses that epichlorohydrin, as an alkylating agent, is "dangerous and powerfully reactive to chemicals" and "combines avidly with tissue." The witness deemed it "certainly possible" that exposure to such a chemical could "depress and destroy the immune system" in humans. Dr. Emmett testified that toxic chemicals cause a form of porphyria called toxic porphyria. He intimated that epichlorohydrin can cause that species of porphyria.

The material safety data sheet[20] issued by Alcolac to the work personnel in compliance with OSHA directive informs that epichlorohydrin, in liquid form, "can be fatal if swallowed," and "[h]igh vapor concentrations can cause death." It informs also that "the results of a recent epidemiology study have been termed 'highly suggestive' that exposure to ECH is a cancer risk to humans." The data sheet describes as the effects of overexposure:

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[19] The qualifications of Dr. Emmett ranged from internal medicine, dermatology, environmental occupational medicine to toxicology - among others in an extensive pedigree.

[20] See APPENDIX I and J for full text of the material safety data sheet issued by the Alcolac supplier, Shell Chemical Company.

"Vapors severely irritating to the eyes, nose and throat. Repeated or prolonged exposure can cause severe and lasting lung, liver and kidney damage and change in lymphocytes [cells in blood and lymph]...Cancer suspect agent." It gives the special precaution: "Notify authorities if any exposure to the general public or environment is threatened by a spill."

Epichlorohydrin was among the toxic compounds spewed about from blow-outs of ruptured disks [as reported by monomer operator Buckner and environmental control technician Holman.] It was also a component of the 2900 gallon and other spills recorded in the Blowers log for year 1981.

ALLYL ALCOHOL

Alcolac used some 416,000 pounds of allyl alcohol per year. It is listed by the EPA as a hazardous waste. It gains entry into the human system by inhalation and is readily absorbed through the skin. Allyl alcohol can have either a chronic or acute effect on humans, according to the concentration of the exposure. The target organs are the liver and kidneys, among other systems, and exposure may cause necrosis. Allyl alcohol also acts as an enzyme and converts other chemicals in the body into even more toxic substances. It has been demonstrated in vitro to alter genetic composition. It was the opinion of Dr. Emmett, Alcolac environmental occupation medicine and toxicology expert, that exposure to allyl alcohol can cause mutagenic effects in humans.

Spills and emissions of allyl alcohol in the monomer building were reported in the evidence. Environmental control tester Holman described an explosion of a monomer reactor which spewed a combination of allyl alcohol, epichlorohydrin and stannic acid. The toxic spill log maintained by environmental control engineer Blowers records a spill of 2900 gallons of AGE stripped brine - a fluid composed of allyl alcohol and epichlorohydrin.

Monomer and surfactant operator Faulconer described a spill of a quart of allyl alcohol whose vapors were allowed to emit into the atmosphere. Allyl alcohol was also a component of the monomer in production at the time of the explosion of September 25, 1981, which blew off a segment of the factory roof and strewed the debris onto private property. It was an observation of one of the numerous WAPORA reports from Bregman to the Alcolac officials that the habitual "sloppy handling" of allyl alcohol in the monomer production process was a cause of the recurrent odors and posed, as well, an environmental hazard.

The material safety data sheet circulated by Alcolac to the plant workers to comply with OSHA directives describes allyl alcohol as a colorless liquid with a sharp odor. In vapor form, it is "highly irritating to the eyes, lungs and damaging to the liver and kidneys." It warns: "Onset of eye irritation may be delayed but can be severe...Absorption through the intact skin can cause injury or death." The sheet concluded with the special precautions: "Handle as flammable liquid and as a poison... Notify authorities if any exposure to the general public or environment is threatened by a spill."

ETHYL ACRYLATE

Alcolac used some 83,000 pounds of ethyl acrylate per year. It is listed by the EPA as a hazardous waste. It is carcinogenic to animals, but present data does not support the expectation that exposure causes cancer in humans. The compound also affects the reproductive process in animals. Dr. Legator, expert in toxicology for the plaintiffs, gave opinion that ethyl acrylate is a mutagenic. The primary portal of entry is inhalation. The chemical is very odorous and extremely irritating. Ethyl acrylate may cause both acute and chronic effects. It is known to cause

convulsions, arrythmias and other strange manifestations of the heart. It also affects the kidneys, heart, lungs, liver and central nervous system.

The wastes from the monomer production process [as a memorandum by Alcolac vice president Bouroff to president Anderson noted] were "saturated with acrylates." It was to allay that source of pollution that Alcolac was required by the abatement agreement with the DNR to install alkaline scrubbers to remove the acrylates from both the liquid and gaseous waste streams from the monomer building reactors. The pronounced odors of ethyl acrylate from the shift in the monomer carbon bed and spills were the subject of report between Alcolac and the DNR.

TOLUENE

Alcolac used some 17,000 pounds of toluene per year. It is an active compound and enters the human system through inhalation and the skin. The compound attacks the respiratory system, central nervous system, the liver, kidneys and heart. It causes arrythmia, tremors, convulsions and other episodes of that ilk. Its effects can be chronic, and - according to Alcolac polyexpert Emmett - presents a "potential risk of cancer" in humans. Exposure to the chemical affects reproduction in animals and - according to an authoritative text - also affects human fertility. It was the opinion of Dr. Zahalsky, immunology expert for plaintiffs, that toluene is toxic to the cells and acts on the immune system. Toluene not only attacks the organ systems of humans, but exposure to the chemical by domestic animals induces a range of effects - such as convulsions, loss of motor control, and even death.

It was this chemical, toluene, which - according to environmental control building technician LeMaire - on occasions overflowed from the sump pits to the exterior and into Shaver Creek. It was this chemical also which spilled outside in such quantity as to cover the adjacent irrigation pond to a depth of

one-half inch. The test of biopond one by the Carnow professional group which found "the footprints of epichlorohydrin" in the waters found toluene also. It was the opinion of toxicologist Legator that spills of toluene and related chemicals exposed to the air evaporate and - depending upon atmospheric conditions - "blow around to the surrounding population."

GLYCIDYL ETHER

Alcolac used some 390,000 pounds of glycidyl ether per year. It is a highly reactive chemical and enters the bodily system through inhalation, the skin or through the mouth - if contained in food or water. Glycidyl ether affects the liver, the kidneys, the respiratory tract and the central nervous system. It causes peripheral neuropathy and manifests in numbness, tingling and leg cramps. Prolonged exposure to the chemical produces chronic effects. Glycidyl ether is known to cause cancer in animals, and, according to Alcolac toxicologist Schwartz, "is possible" as a cause of cancer in humans.

Toxicologist Legator, in response to a hypothesis posed from the evidence of ninety residents within the Alcolac environs that from 50% to 60% of them experience continuous tingling and numbness of the extremities, equated that incidence with "walking down the street and seeing seven people who are eight feet tall."

CYCLOHEXANE

Alcolac also used cyclohexane in the monomer process. [The evidence does not disclose the quantity.] It is listed as a hazardous waste by the EPA. The portal of entry is inhalation and the target organs are the respiratory and the central nervous systems. It causes erratic blood pressure, sudden nosebleeds, lethargy, depression and vascular disorders.

In addition to the six chemicals already tested for toxic effect - epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, glycidyl ether and cyclohexane - as explained by the

testimony of toxicologists Legator, Schwartz, Emmett and Carnow, the Alcolac evidence identified other chemicals, also of known effect, involved in the production process. Among them were dimethyl sulfate, hydrogen sulfide, methyl chloride and allyl methacrylate.

DIMETHYL SULFATE

That chemical, Alcolac toxicologists Schwartz and Emmett agreed, enters the bodily system through inhalation and the skin. The kidneys, liver, and the central nervous and respiratory systems are the target organs. If the exposure is persistent enough and the dosage sufficient, the effects of the chemical can be chronic. It is a carcinogen in animals and, according to Dr. Emmett, carries risks of cancer to man. Dimethyl sulfate, immunologist Zahalsky explained, is an alkylating agent: it adds new chemical groups into the compound. As in the case of epichlorohydrin [and some of the other chemicals], absorption into the human system operates to alter the DNA and to kill normal cells. In the terminology of immunologist Zahalsky, dimethyl sulfate attaches to cells and induces the genetic code sequence "to go haywire." It was an opinion confirmed by Alcolac immunologist Stechschulte.

The material safety data sheet circulated by Alcolac to the plant workers to comply with the requirements of OSHA warned: "Extremely hazardous liquid and vapor. Causes severe burns. Lung injury and burns may be delayed. May cause cancer [based on tests with laboratory animals]."

HYDROGEN SULFIDE

"Hydrogen sulfide is deadlier than cyanide," Dr. Carnow explained. This compound is absorbed into the bodily system through inhalation and the odor of rotten eggs attends its presence. The odor of rotten eggs was a recurrent emission from the bioponds. The cause of these odors Dr. Carnow attributed to the

production of hydrogen sulfide from the overload of the bioponds with toxic chemicals. It was the recurrent rotten egg odor of hydrogen sulfide in the sludge of the overused bioponds that Dr. Reid detected during his tests and examinations for WAPORA. It was also hydrogen sulfide DNR official Nikkila detected during one of his investigations of the bioponds. He attributed that emission to insufficient aerators or to overloaded use.

The organs affected by exposure to this compound are the liver, the kidneys, the respiratory and central nervous systems. It was the opinion of Alcolac toxicologist Schwartz that hydrogen sulfide produces chronic effects if the dosage is adequate of a prolonged exposure. In such circumstances, damage to the liver could result in damage to the porphyrin metabolism. Dr. Carnow elaborated: In relatively low concentrations, hydrogen sulfide causes dizziness, vomiting and severe headaches. In higher concentrations, it causes brain damage, respiratory paralysis, and death.

METHYLENE CHLORIDE

It was a chemical found in the biopond waters in the tests performed for the plaintiffs by the Carnow professional group. It was among the toxic wastes [according to monomer operator Faulconer] stored in drums, kept along the Alcolac driveway and crushed by truck traffic so that the highly volatile compound was permitted to emit into the atmosphere. It was among the toxic waste remnants, according to Faulconer, Alcolac personnel discarded from tankers before replenishing them. Methylene chloride enters the human system through inhalation and the skin. In a sufficient dose it affects the liver [and hence porphyrin metabolism], the heart, and central nervous and respiratory systems. It was the opinion of Alcolac toxicologist Schwartz that in adequate and prolonged dosage, methylene chloride may cause cancer and mutations in humans and depress the immune system.

HYDROCHLORIC ACID

Hydrochloric acid - according to WAPORA president Bregman, was a product of the misuse of the liquid incinerator in the environmental control building. Inhalation [in vapor form as hydrogen chloride] is the principal portal of entry. The vapor becomes an acid when dissolved in water and in that form burns through the skin. In large enough dosage, Alcolac toxicologist Schwartz explained, hydrochloric acid can cause damage to multiple organs: the respiratory and central nervous systems, the liver, and the kidneys. It was the opinion of Alcolac toxicologist Emmett [in apparent disagreement with Alcolac toxicologist Schwartz] that the compound can induce chronic effects.

ALLYL METHACRYLATE

Alcolac produced allyl methacrylate in the monomer process. The material safety data sheet circulated by Alcolac to the workers describes the effects of overexposure: "Product may be fatal if inhaled, ingested or absorbed through skin. Irritation of eyes, nose, throat; dermatitis; narcosis." A spill of 15 gallons of allyl methacrylate was recorded by Blowers in the log on July 29, 1981, from a tank overflow.

There was evidence [already recounted] that from the virtual onset of the Alcolac operations in 1978, these chemicals or their pyrolysis products were regularly emitted into the atmosphere through the defective liquid incinerator, the vented spills, or discharges - unfiltered and still toxic - into the bioponds which mixed with the soap refuse and in that form were borne away into the ambient air. The function of the liquid incinerator, as explained, was to destroy by heat the organic matter of the toxic chemical waste from the monomer process so that liquid would emerge as wastewater free of contamination and flow into the biopond. When the incinerator malfunctioned, however, the organic matter in the toxic liquid waste remained unconsumed or only

partially consumed. When the waste was not consumed, it was emitted into the atmosphere unchanged and still toxic. When the waste was partially consumed, it was emitted as chemical fractions of the toxic waste, often with multiplied toxic effect.

The remnants of these unconsumed wastes retained in the liquid, and their pyrolysis products, moreover, then flowed into the biopond to mix with the toxic wastes periodically pumped around the filtration system and into the biopond. The effect of such a potpourri [in the terminology of Dr. Carnow] was "a witch's brew," a "chemistry set that is unbelievable [of] extraordinarily active chemicals, alkylating agents, like epichlorohydrin. [Y]ou put them all in one place and there is no way of knowing the extraordinary number of toxic agents that you might produce in such a soup." It was significant, Dr. Carnow explained, that the chemicals used and emitted in the monomer production process - epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, glycidyl ether and cyclohexene - attack common organ systems: the liver, the immune system and the central nervous system. He described the mode and effects of concerted assault:

> They are extremely active. That's why they are so irritating to the eye and the nose, because they attack protein . . . They attack and can even coagulate protein, and when they get into the body, they head for organs, and . . . can cause very severe damage, but again in low concentrations, they eat away at an organ. They cause damage, and if you have more than one . . . you may have either what we call an additive effect, that's where one and one is two, or you may have what we call a synergistic effect, and that's where two chemicals have more than an additive effect. That's when two and two is six, or ten, or something like that, and that makes it even worse. You have the worst of all possible situations when you have chronic, recurrent exposure, you have multiple chemicals that are highly reactive, you have multiple portals of entry, and you have these chemicals acting together to attack major organ systems.

IV

Residents in the Environs of Alcolac

A

The Plaintiffs-Witnesses

Alcolac is located on an industrial site northeast of the Sedalia limits in the midst of land still used for agricultural purposes. The industrial area of Sedalia proper lies to the southwest of Alcolac within the city boundary. The thirty-one plaintiffs, composed of twelve family groups, all resided within one and one-half miles of Alcolac - most of them much closer.[21] All but one of the family groups had established residence there before the advent of Alcolac.[22] The ages of the plaintiffs range from 75 years [Virgil Bradley] to 11 years [Amber Cross]. It was the uniform testimony of all the plaintiffs that the onset of the foul and noxious odors, fumes, smoke, vapors and emissions they experienced coincided with the commencement of operations by Alcolac. Storms of foam from the bioponds were borne onto their land [23] and onto Little Shaver Creek and transported throughj other properties.[24] It was the uniform testimony of the plaintiffs that exposure to these Alcolac emissions caused them symptoms - most commonly and recurrently - eye, nose, throat and skin irritations, nausea, vomiting, numbness and tingling of the limbs, heart irregularity, respiratory difficulty, and other afflictions. The odors were described variously but recurrently as those of rotten

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[21] See Appendix B. The dark circles indicate the residences of the several plaintiff families. The heavy, dark line in approximately the middle of the Appendix marks the bounds of the Alcolac site.

[22] The plaintiff Gehlken purchased the residence before Alcolac commenced operations in May of 1978, but took up actual residence thereafter, in July of 1978.

[23] See APPENDIX C.

[24] See APPENDIX D.

eggs, sulphur, lacquer, ammonia, of sweetness, and of other familiar scents. It was the uniform testimony of the plaintiffs also that they had experienced no noticeable emissions or impairment of health from the operations of other enterprises in the locale, in place since before the advent of Alcolac.[25] There were innumerable reports of these annoyances, intrusions and physical effects to Alcolac, the DNR, and governmental officials at every level, but to no avail.

The frequency and intensity of the atmospheric emissions from Alcolac, these witnesses agreed, depended upon the direction or calm of the wind. There was expert meteorological testimony, derived from statistical observations taken at Whiteman Air Force Base near Sedalia, that the wind blows from the south 12.5 percent of the time, blows from the south-southwest 8.5 percent of the time, from the north 8.5 percent of the time and remains calm [that is, blows at less than 1 mile per hour] 13.5 percent of the time. The wind is light and variable 47% of the time. That latter statistic, the expert concluded, indicated "a very excellent opportunity for the people in the vicinity of the plant to detect odors a large percentage of the time."

The members of each plaintiff family group testified to the recurrent incidences of exposure to the Alcolac emissions and the physical symptoms they induced, to their life modes and habits, and to other personal data which relate to the expert assessment of the etiology and causation of their complaints of environmental injury. This history data was of the same kind elicited from each of the plaintiffs upon clinical examination and, along

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The enterprises located in the industrial zone of Sedalia to which Alcolac alludes as a source of emissions and discomfort to the plaintiffs include, most conspicuously, the Missouri Pacific Railroad yards, Missouri Pressed Metals, DeLong, Quality Fiberglass, among others.

with the results of physical examination and laboratory tests, were significant elements in the derivation of environmental medicine diagnoses by their physicians as well as those of Alcolac.[26]

## B

## The Non-Litigant Witnesses

### 1.

### For the Plaintiffs

There was testimony, for the plaintiffs, from more than fifty other witnesses, all nonlitigants also resident in the environs, that the Alcolac emissions were causes of annoyance and physical complaint. The locations of their residences ranged from very next to the Alcolac plant to sites more than three miles distant.[27] Their occupations were as diverse as their ages. The current of that testimony comported with the testimony of the plaintiffs: the Alcolac operations gave off emissions in the form of vapors, odors, smoke, fumes, foam and residues which physically affected their persons. The odors and emissions were persistent and recurred day and night. Black smoke belched from the Alcolac stack even in the early mornings. A white residue covered the grass and other vegetation and was an irritation to the skin and exposed areas of the body. Suds and foam from the Alcolac bio-ponds alighted on their property, even though several miles distant.

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[26]The witness responses are found in gist in APPENDIX E.

[27]See Appendix B. The residences of the nonplaintiff witnesses are marked by light colored circles.

They [as did the plaintiffs] described the odors as scents of rotten eggs, lacquer, sickening sweetness, ammonia, sulphur - and the like. They [as did the plaintiffs] described the consequences of exposure to these discharges as irritations to the nose, throat and eyes, matted eyes, redness and watering of the eyes, kidney infections, skin rashes, headaches, cramps and charley horses in the calves and legs, numbness and tingling in the limbs, nausea, fatigue, respiratory difficulty and irregularity of heartbeat. The more youthful females also complained of interrupted and abnormal menstrual cycles. The symptoms, as to most of them, persist. As to several of these nonlitigants who removed from the environs of Alcolac, the symptoms disappeared. They [as did the plaintiffs who kept gardens and animals] complained that the emissions, especially in the form of a white powder residue, afflicted the vegetation so that whole stands of decorative, orchard trees and vegetable plants were stunted. Their domestic animals and pets also died - and unaccountably and without sign of predation. They included cultivated husbandry such as bee swarms, cattle and chickens as well as domestic pets, such as parakeets, dogs and cats. There was also evidence by a non-plaintiff witness that Little Shaver Creek [which traversed the property], the source of the drinking water for the cattle, was so tainted by foam from Alcolac as to kill the fish and cause the cattle meat to stink and become inedible.

It was the testimony of these witnesses [as of the plaintiffs] - a number of whom resided for years adjacent to the Missouri Pacific Railroad yards, Missouri Pressed Metals, DeLong Welding and other plants - that not until Alcolac commenced operations were any odors or other emissions palpable, and not until Alcolac were any ill effects to their persons or plants and animals experienced. A very few of them noticed an occasional odor from Quality Fiberglass, but it was fleeting and of a different

quality. The odors and emissions from Alcolac were distinctive and recurrent. It was the testimony of those among them whose employment brought them onto the Alcolac premises for periods of time - such as construction workers - that the odors and emissions they detected from their residences were of the same kind they experienced while in the Alcolac plant. It was the testimony of all these nonlitigant witnesses that their symptoms, complaints and changes of health came on after the Alcolac operations began in May of 1978, and not experienced before.

The nonlitigant witnesses for the plaintiffs also included public officials, some of whom lived within the environs and others who did not, but all of whom frequently - even repeatedly - were called to the vicinity of Alcolac and onto the premises, day and night, in response to citizen complaints of odors and other emissions. The experiences of emissions from Alcolac and the symptoms from those exposures were congruent with those described by the plaintiff witnesses as well as the other nonlitigant witnesses for the plaintiffs. Odors were emitted, smoke from the stack, dense haze, and suds and foam from the bioponds. The odors were described, variously, as "intense," "terrific," "bad" - of the sulphur type, of rotten eggs, septic or paint thinner. The odors were the same off the Alcolac premises as they had detected in the plant, and were noticeable from several miles away. The foam and suds from the bioponds came in profusion - a phenomenon Senator Mathewson described as: "[L]ike Lawrence Welk had turned loose the bubble machine." The vapors sometimes emitted as "a very fine mist" and burned exposed skin. County Court member Jones described the effects to exposure as irritated and watery eyes and headaches. Sgt. Rice of the Sedalia police department [designated by the DNR to respond to citizen complaints] experienced irritation to the mucous membrane from exposure to the Alcolac emissions, as well as irritation to the nose and throat,

watering eyes and upset stomach. Environmental Quality Control Commissioner Judy Berenyi suffered headaches, nausea, and a "stinging skin" even from a momentary exposure during the course of her investigations of citizen complaints." The public official witnesses agreed that there had been no citizen complaints of odors or emissions or physical complaints from the operation of the other plants in the vicinity - such as the Missouri Pacific Railroad yards, Missouri Pressed Metal, DeLong Welding.

The plaintiffs presented other nonlitigant witnesses, employees of Alcolac, whose descriptions of the emissions, their odors, as well as the physical symptoms induced by exposure, coincided with those of the plaintiffs and their witnesses. These employee witnesses - Pace, Buckner, Faulconer and LeMaire - identified the recurrent chemical spills, efflux of contaminated wastewater into the bioponds, monomer reactor explosions, liquid incinerator and acid scrubber malfunctions and, simply, careless management of the chemicals as the sources of the Alcolac emissions and odors. The detection of these emissions as odors of sulphur, rotten eggs, sweetness -- and a variety of other descriptions - was explained by the characteristics of the chemicals used in the particular phase of production. They testified that exposure to these chemicals and emissions induced in them [as in the plaintiffs and the other witnesses] a burning and watering of the eyes, an irritation to the skin, nose and lungs, cramps in the legs and feet, and numbness and tingling in the extremities.

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For the Defendant

Alcolac also presented nonlitigant witnesses, a dozen or so, who resided in the environs or had occasion to come onto the plant premises. They included farmers, businessmen and public

officials, as well as employees. A number of them resided adjacent to some of the plaintiffs. Several of the witnesses conducted regular contract work with Alcolac, and a number of the residents had family members employed by Alcolac. Witness Knaus, a farmer, sharecropped Alcolac land. Witness Sneed, also a farmer, cultivated pollen on Alcolac property. There were several among them unrelated to Alcolac by business or personal interest.

There were some who experienced odors on the residence sites, or when they passed by Alcolac, or when they went upon the plant premises. There were some who discerned none. There were some who had seen foam from Alcolac alight on their property. Those who detected odors described them variously: as rotting eggs, pleasantly sweet, or as new plastic. It was the consistent response to inquiry that the emissions caused no ill effect to their persons. Those who kept gardens, crops and livestock all responded that neither the productivity nor the quality was affected. The well-being of their pets was not impaired.

There were other acknowledgments on cross-examination, however. Farmer Reine [who lived near the plaintiffs Turley] answered that some of his cows miscarried for reasons the veterinarian could not explain. He responded also that the children suffer "terrible headaches," as does the wife, that she experiences arrythmia and palpitations of the heart, has developed cramps on the calves of the legs, and suffers from an irregular menstrual cycle. Pettis County Sheriff Starke responded that exposure to Alcolac odors brought on a headache and a "burning, peppery sensation in back of the throat." Resident Vicki McFall answered that a drive by the Alcolac plant "burns your nose.: The regular employment medical examinations, Alcolac chemical operator Henderson acknowledged, cite his complaint of numbness and tingling of the hands or feet. The medical examination reports of Alcolac plant chemist Richardson note the development of abnormal

globulins and hemoglobin. The medical examination reports of Alcolac quality control technician Theresa Cecil disclosed two immunity panel abnormalities.

V.

The Medical and Scientific Evidence.

A.

The Family Practitioner
Dr. Donald J. Allcorn

A number of the plaintiffs consulted Dr. Donald J. Allcorn as personal physician, both for treatment of symptoms they attributed to the Alcolac emissions, and for other reasons of health. Dr. Allcorn, board certified in family practice, maintains an office in Sedalia. Two among them - Clarence Elam and Dan Pryor - were treated by Dr. Allcorn for injury from mishaps unrelated to the Alcolac operation. The other seven - Jacqueline Berry, Virgil Bradley, Dorothy Bradley, Betty Elam, Linda Elam Sanders, Joyce Pryor and her daughter Amber Cross - consulted with Dr. Allcorn for complaints attributed to Alcolac chemicals, as well as for other conditions.

Dr. Allcorn was presented by Alcolac. He gave opinion that toxic chemicals can cause many of the conditions and symptoms the several patients displayed, but that they did not - "other than [as to Mr. Bradley, and presumably, the others with like symptoms] those related as possible irritation of his airways due to odors." He acknowledged both on direct and on cross-examination that exposure to toxic chemicals can cause chest congestion, urinary tract infection, nose and sinus irritation, skin rashes, abdominal pain, tingling limbs, conjunctivitis, fatigue, respiratory infection, sore throat and dizziness - the other symptoms and complaints displayed by these several patients. He concluded, however, that none of these symptoms were the result of toxic

chemical poisoning, but could be explained by other causes. In the case of Virgil and Dorothy Bradley [75 years old], he implicitly attributed the complaints to their condition of age. In the case of Betty Elam, he attributed the condition of emphysema to cigarette use. Although the witness attributed the other complaints of the patients to causes other than exposure to toxic chemicals, he did not identify those alternatives.

The opinions rendered by Dr. Allcorn were those of a physician certified in family practice. He readily acknowledged that he had neither special training nor qualification in occupational medicine, preventive medicine, toxicology or immunology. Nor had he dealt with the toxic chemicals used in the monomer production and emission processes - such as epichlorohydrin, allyl alcohol, dimethyl sulfate, toluene and the others.

B.

The Immunologists

The testimony and opinions of the immunologists were based on three sets of laboratory procedures performed between August of 1984 and July of 1985, to test the immune system functions of the several plaintiffs. The plaintiffs presented the testimony and opinion of Arthur C. Zahalsky, professor of microbiology and certified research immunologist. Alcolac presented Daniel J. Stechschulte, certified medical internist and certified clinical immunologist.

The first immune panel was conducted by the Bioscience Laboratory in Chicago [August of 1984] as an incident of the clinical examinations and diagnoses of the plaintiffs by the

Carnow Associates, their experts. All thirty-one[28] plaintiffs presented themselves for this series. The second immune panel tests were conducted by the Kansas University Internal Medical Center [December of 1984] at the instance of Alcolac. The protocol for that series of tests - as defined by Alcolac counsel and internal medicine expert Kerby, a professor at the University of Kansas Medical Center - excluded ten of the plaintiffs.[29] It was the surmise of Alcolac expert Stechschulte that the protocol did not encompass the other ten because: "[T]he information that was available . . . didn't dictate that these individuals had abnormalities in their immune systems." The third immune panel was conducted by the Midwest Organ Bank and Wheeler Laboratory in Kansas City [July of 1985] at the instance of immunologist Zahalsky. He was prompted to that initiative by a deemed inadequacy of the KU Internal Medical laboratory test results and because the pervasive abnormalities disclosed by the Bioscience immune tests suggested the need for more particular information of the cell compartments. He explained that the KU laboratory blood analysis tests were inadequate because they expressed results in percentages rather than as within or without normal range references - as is the common practice - with the consequence that the counts of the various cell populations could not be determinated nor the normality of those counts. The KU procedure was all the more wanting, he explained, because that protocol did not include

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[28]Edward Gehlken, an original plaintiff, was disabled from a series of strokes and did not present himself for any of the examinations, nor for trial testimony. The judgment directed against his claim by the trial court is not on appeal.

[29]Malva Gehlken, John Phillips, Daniel Pryor, Joyce Pryor, Linda Elam Sanders, Arnold Sommers, Kay Turley, Lyle Turley, Amber Cross and Dorothy Bradley. Edward Gehlken was also excluded by the definition of the protocol, but he is not accounted a party for the purpose of opinion.

all of the plaintiffs. It was for those reasons that immunologist Zahalsky arranged for the third immune panel procedure at the Midwest Organ Bank and Wheeler Laboratory in July of 1985. Twenty-four of the plaintiffs were tested. Mrs. Landon by then was under a regimen of cobalt treatment for cancer, and so was disqualified for the tests. The six members of the Turley family were unavailable. The evaluation rendered by Zahalsky as to the Turley family members rested on the tests reported by the Biosciences Laboratory in August of 1984. The opinion as to Landon rested on both the Biosciences and the KU immune panel results.

Arthur C. Zahalsky, PhD

The witness defined immunology as the study of the components of the immune system of man and animals - the cells that compose the system, the appearance of those cells, the origin of those cells, the presence of those cells in the blood, and the balance of those cells. He likened the immune system to "an internal watchdog or searchlight" which surveils the cells and expels those components which are not "okay," and thus restores the homeostasis - the normal state of immunologic balance. The component extruded from the body by this process, the witness explained, might be the result of a mutational effect caused by the activity of a bacterium, or a virus, or of a toxic chemical. An immune dysfunction, accordingly, is the imbalance and dysfunction of the cells of the blood. The consequence of dysfunction is that the "internal searchlight" becomes dim so that harmful substances may appear in the body which escape recognition, and hence elimination. One result of dysfunction may be neoplastic disease - a condition of cancer. Another result, among the many others, may be the inability of the body to fight infection. The immune system function is determined by the genetic code of the person

- none of them exactly alike. The efficiency of the genetic system depends upon the stresses imposed by the mode of life and habits adopted by the person: smoking, the consumption of excessive alcohol, abuse of drugs all stress the immune system. That is to say, they affect how the genetic code expresses itself.

The immunologist noted that the immune systems of the plaintiffs and of those nonlitigant residents of the environs of Alcolac shared a common insult: the prolonged exposure to the toxic chemicals in the environment in which they lived. He undertook to explain the consequences of the exposure to the toxic chemicals identified with the Alcolac production process in terms of the function of the human immune system. The immune system is not a specific organ but is distributed in the bone marrow, the thymus, the spleen, and in the lymph nodes throughout the body. In terms of function, the immune system has two divisions. One function is the production of antibody cells which are dumped into the blood and circulate to all parts of the body. These antibodies, called B-cells, derive from the bone marrow and spleen and fight off infection. Another function of the immune system is to "search and destroy," and is the "cell mediated" arm of the system. Those cells include the T-cells, so-called because they derive from the thymus gland, although they derive also from the lymph nodes and spleen. The T-cells exhibit distinctive characteristics and are of four different types. They remember previous insults and challenges, they live long, and have the capacity to expand. The four types of T-cell populations are the helpers,

suppressors, cytotoxic and natural killers. The helpers cooperate with the B-cells to produce antibodies. The suppressors work in balance with the helpers to avoid indiscriminate and undirected responses. The cytotoxic cells are also suppressors and can attack and destroy transformed [neoplastic] cells with or without an antibody. The natural killer cells can also attack and destroy without the help of an antibody.

An imbalance of the helper populations of T-cells [technically designated as T/4], when induced by stresses and insults of toxic chemicals, is a condition of immune system dysfunction properly designated: chemically induced AIDS. In common AIDS, it is the virus [rather than the chemicals] which attacks the helper [T/4] cells and so induces immune system dysfunction.[30] In either case, whether the imbalance is induced by toxic chemicals or by virus, the result is that much of the T/4 cell population is depressed - "functionally wiped out." Thus, the balance in the bloodstream "just goes completely out of whack" and the production of T-cells cannot keep up with the demand. The consequence, among other dysfunctions, is a progressive erosion in the ability of the person to fight infection. In the case of viral AIDS, the customary course of infection is in the lungs, which eventually leads to respiratory collapse, and death. In the case of chemical AIDS, the progressive erosion of the immune system may induce neoplastic disease - and cancer. Chemically induced AIDS, the witness testified, is a phenomenon supported in the medical and immunological literature, and is a subject of research at the National Institute of Environmental Sciences.

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[30]The viral version of AIDS sometimes goes by the designation HTLV - which stands for "Human T-cell leukemia virus."

It was the opinion of immunologist Zahalsky that the condition of chemically induced AIDS could be brought on by exposure to the chemicals regularly used and produced in the Alcolac monomer process. Epichlorohydrin, toluene and dimethyl sulfate, among the others, are toxic to cells and act to depress the immune system. Dimethyl sulfate is also an alkylating agent which "sticks" to cells so that the genetic code sequence "goes haywire," renders the cells toxic and suppresses the immune system.[31] The witness gave opinion that exposure to a "chemical soup" of epichlorohydrin, toluene, dimethyl sulfate, among the others emitted into the Alcolac bioponds over a span of seven years and then carried into the air by suds and vapors, could cause a depression of the immune system to persons in the environs. The actions of these chemicals often induce symptoms which mimic actual diseases, the witness explained, so that an un trained local physician will not recognize that the complaints and symptoms actually bespeak a depressed immune system toxically induced.

Zahalsky evaluated the immune system of each of the thirty-one plaintiffs. The witness, a research immunologist but not a clinician, rested the assessments on the laboratory and test data gathered in the immune panel reports from the Bioscience, KU and Midwest/Wheeler laboratories. The witness evaluated the immune system of each plaintiff according to whether the result for each value tested was within or without a normal reference range.[32] Thus, by the self-evident standard adopted by the expert

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[31]Ample toxicological profiles of each of the most toxic chemicals prominently involved in the Alcolac monomer production process were rendered by toxicologists Legator, Schwartz and other experts, as already noted.

[32]The common practice [Zahalsky explained] is for the normal reference range for each test of the various blood cell populations to be printed on the face of every test report form. The cell population values of particular interest to an immune deficiency evaluation are the total T cells, the count of helper T cells [T/4], the count of suppressor T cells [T/8], and the helper/

witness, an abnormality is a test value that falls outside the normal reference range. Also, by that self-evident standard, "abnormality is not good." Hence, the conclusion of the witness: "a laboratory report that fails to list the reference ranges is invalid by definition." It was, as we note, the perceived unreliability of the KU test results expressed in terms of percentages rather than in terms of reference ranges which prompted witness Zahalsky to recommend that the subjects undergo a third test regimen at the Midwest/Wheeler laboratory.

The summary of test results from the Bioscience Laboratory noted at least one abnormality, and as many as eight, in the immune system of every plaintiff.

The test results for every plaintiff from the KU laboratory, expressed in terms of percentages, were interpolated by the witness through the reference ranges of the Midwest/Wheeler forms into determinations of normal, abnormal and marginal. The KU protocol [as did the Midwest/Wheeler tests] encompassed total T cells, helper cells [T/4], suppressor cells [T/8], natural killer cells and other populations. Zahalsky deemed that the most significant test result components were the helper cell [T/4] and

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suppressor ratio, the mature B cell count and the natural killer cell count - among others. The normal range for each of these values - as is the common laboratory practice - is printed on the face of each of the Midwest/Wheeler test report forms. These ranges are derived from the most current immunologic literature and from other authoritative sources - such as Eli Lilly "Medifacts" and the Smith-Kline Labs. The witness verified that the reference ranges adopted by the Midwest/Wheeler report form were "right in line" with the norms published in the professional reference source, Diagnostic Immunology.

The results of the KU immune panels, Zahalsky testified, not only expressed results in terms of percentages rather in terms of normal reference ranges, but they also failed to report the results in terms of absolute numbers. Thus, as the Midwest/Wheeler test for plaintiff Carl Berry reports, the total T cell count for each unit of blood was 1,045 cells. That datum, as well as other such values essential to a determination of immune deficiency, is lacking in the KU panel of tests.

suppressor cell [T/8] counts. The test result associated with the chemical AIDS condition, Zahalsky noted, was an abnormal helper cell [T/4] count. In the case of plaintiff Carl Berry, the KU test disclosed a T/4 count fully five percentage points above the average - an abnormality. In the case of Virgil Bradley, the transposed test results disclosed four abnormalities. Of the twenty-one plaintiffs tested under the KU protocol, the transposed results established that the blood cell population of only one - Joy Sommers, age 15 - displayed no immune system abnormality. The composite tests disclosed 39 abnormalities of the T cells distributed among the 24 persons tested.

The results from the Midwest/Wheeler Laboratory established that 19 of the 24 plaintiffs who presented themselves for tests suffered from dysfunction of the immune system. The witness explained that since the T cell population, as an absolute number, changes with age, to enable valid analysis of dysfunction, he segregated the plaintiffs into three age groups -the young, the intermediates, and the older. The tests of fourteen among the plaintiff groups disclosed the condition of hypo-dysfunction - that is, a depression in the total number of T cells. The tests of five among them disclosed the condition of hyper-dysfunction - that is, an elevation in the total number of T cells. Thus, 79% of the plaintiffs tested by the Midwest/Wheeler Laboratory displayed an immune dysfunction of the T cell compartment. The immunologist deemed the finding "tremendously statistically significant." To this number the witness then added the six members of the Turley family group who were not available for the Midwest/Wheeler regimen and Mary Landon by then under treatment for cancer. All of them were earlier tested at the Bioscience laboratory and demonstrated immune dysfunctions. Thus, the combined Bioscience-Midwest/Wheeler immune panel tests established that 26

out of the 31 plaintiffs, or 84% of the total number, demonstrated an immune dysfunction. The witness described that statistic "an astounding value."

The Midwest/Wheeler immune panel tests included a newly available procedure administered at the direction of immunologist Zahalsky. It was for the T cell monoclonal antibody, HNK-1. That antibody not only detects the natural killers of the T/4 population but also suppressors not found in that population. Ten out of the 24 plaintiffs had significantly elevated values in HNK-1, and one person had a reduced count. Four among the plaintiffs tested[33] not only displayed elevated levels of HNK-1, but also elevated levels of T/8 suppressor cells. Those values indicated to the expert a gross distortion in the ratio between the helper cells and the suppressor cells - an indication that the immune system balance is "out of whack." They already suffer severe immune dysfunction, the condition of chemically induced AIDS. Eight others[34] already suffer moderate immune dysfunction, although not yet the condition of chemically induced AIDS. The changes in the immune system panels, however, are progressive so that the dysfunctions will develop to the AIDS condition "somewhere down the line."

The dysfunctions of the immune panels as trends in progress were demonstrated by the results from the Bioscience and the Midwest/Wheeler laboratories - the two tests the witness deemed reliable. The Bioscience test conducted in August of 1984 reported one abnormality of the immune system for Arnold Sommers. The Midwest/Wheeler test conducted in July of 1985 listed two abnormalities. The progression for Dorothy Bradley listed by

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[33]Virgil Bradley, Dorothy Bradley, Malva Gehlken and Dainie Landon.

[34]Amber Cross, Gwendolyn Lawrence, Joyce Pryor, John Phillips, Arnold Sommers, Joyce Sommers, Joy Sommers and Genevieve Withers.

those tests within that interim were from one abnormality to three, and for Virgil Bradley, from one abnormality to five. He noted also that the immune panel of findings of laboratory tests taken of Mary Landon before her cancer condition and cobalt therapy verify that she had a severely depressed immune system. It was the opinion of immunologist Zahalsky, moreover, that the conditions of the other nineteen plaintiffs who then manifested neither severe nor moderate immune system dysfunction would progress into proliferated abnormalities.

Zahalsky concluded with the opinion that all thirty-one plaintiffs were suffering from systemic, progressive chemical intoxication. The witness gave opinion that the condition is the consequence of exposure of the plaintiffs in their home environment to toxic chemicals which have induced changes in their immune system. That injury has dampened the immune system so that the plaintiffs will become subject to a variety of diseases, neoplastic disease [cancer] included. The findings already suggest the possibility of leukemia. The expert would not attempt the clinical prognosis as to "just how sick this group of people is at the present time and is likely to become." That was a matter for a competent clinician - such as Dr. Carnow, an occupational medicine expert. He did conclude, however, that since the condition of dysfunction was progressive, each of the plaintiffs should have annual laboratory tests for the rest of their lives, and bi-annual tests for the four who already exhibit severe dysfunction. The cost for each such laboratory service is $1000 per person, at the current rate.

Daniel J. Stechschulte, M.D.

Alcolac presented Daniel J. Stechschulte, board certified immunologist and internist, and Director of the Division of Allergy, Clinical Immunology and Rheumatology at the University of

Kansas Medical Center. He treats patients at the Medical Center, as well as teaches, but has never treated anyone for a toxic chemical complaint or conducted research on that subject.

The witness described the immune system in the same terms earlier used by Dr. Zahalsky. His response as a physician to a patient who presents a possible immune system problem is to examine the patient, take a history and review the available information. Symptoms and findings of recurrent infections, hypersensitivity reactions or auto-immune diseases[35] suggest immune system dysfunction. A laboratory test may be the adjunct of a more precise evaluation, and the selection of the test depends upon the nature of the symptoms and findings. They help to identify the arm of the immune system involved, and, accordingly, the antibodies of that arm are tested. A test result which does not fall within the reference range - that is to say, an abnormality - does not ipso facto signify immune system disease. The abnormality, rather, may be the effect of a variable exogenous to the immune system - such as the state of nutrition -which stunts its normal operation. Thus, the clinician interrelates the test abnormalities to the age, gender, nutrition, mode of life of the patient, or other variable to determine whether the variables account for the test abnormalities or if some other cause explains the immune system dysfunction. Thus, the witness explained, the end of immunologic evaluation is not a count of cells, but how well the cells function as elements of the immune system, whatever their count.

Viral infections can alter the immune system. Certain toxic chemicals can also alter the immune system. The witness

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[35]Auto-immunity describes the condition of a misdirected immune system which reacts against the person adversely and destroys the tissues.

acknowledged the condition called chemically induced immune dis-regulation. He discountenanced the term chemically induced AIDS to describe that condition of immune dysfunction, however, because "that common usage [AIDS] now refers to individuals that are infected with the human T-cell leukemia virus, Type III."

Dr. Stechschulte was consulted as to the immunologic tests to be administered to the plaintiffs under the KU protocol. To inform that advice, Dr. Stechschulte reviewed the available test and clinical records of the several plaintiffs from the Carnow clinic, as well as numerous medical records from other sources. The Midwest/Wheeler immune panel tests had not yet been conducted, and so those results were not available to Dr. Stechschulte. He evaluated these sources for findings of hypersensitivity reactions, recurrent infections or auto-immune disease [indicia of immune system dysfunction], but saw no "predominance of symptoms in any of these areas" - except for Mary Landon. He nevertheless advised certain immune panel tests for twenty-one of the plaintiffs. The decision to exclude ten of the plaintiffs from the tests under the protocol was prompted by the results already derived from the tests administered through the Carnow clinic. Dr. Stechschulte accepted those results as "reliable" and "authentic." The information they yielded, he explained, "didn't dictate that these individuals had abnormalities in their immune systems," and hence the need for further tests was discounted. The selection of plaintiffs for tests, Dr. Stechschulte explained, was by random method, and included "patients that were identified as having immune dysfunction" as well as those the records suggested "were perfectly normal."

The immune panel tests were administered by Dr. Bodensteiner of the Department of Medicine at the University of Kansas Medical Center. The regimen adopted, the witness explained, was

to test function and not merely to count cells.[36] The functional test elicits how the immune system responds when it is exposed to a particular antigen or organism. The test, actually conducted in the test tube and not in the human body, simulates the response of the immune system to an infection stimulation. The tests administered through the Carnow clinic, the witness acknowledged, were both functional as well as cell counts and were more numerous than the single function test performed under the KU protocol.[37] The immune deficiency tests conducted by Midwest/Wheeler, on the other hand, were expressed in cell counts but did not measure functional response. Immune deficiency tests measured exclusively by cell counts, the witness suggested, assume a relationship between a normal number of cells and normal function of those cells.

The evaluator of the KU test results - immunologist Stechschulte - was in any event aware of the various reference ranges and undertook, within a reasonable medical certainty, to interpret the KU results as to each of the plaintiffs subject to the protocol. With the exception of Mary Landon, the evaluator concluded, the clinical history of the plaintiffs examined suggested that "their immune system was functioning normally." There were some, he acknowledged, "that have laboratory tests that fall outside the arbitrarily defined normal ranges." Others displayed

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[36]The testimony of witness Stechschulte intimates that the KU test results were formulated without reference ranges because Dr. Bodensteiner had not yet "put in a cell sorter" - the apparatus used by Midwest/Wheeler for cell counts. In fact, as immunologist Stechschulte described the test result procedure, Dr. Bodensteiner relied on the Midwest/Wheeler test counts to compare and validate his own results: "He [Bodenheimer] basically took individuals and took paired samples, and sent one to Midwest Organ Bank and sent the other to his machine, and then compared the results when they came back."

[37]The functional tests were in the form of mitogen challenges - [a procedure more fully discussed in the course of opinion] which assesses the ability of the lymphocyte component of the immune system to defend against infection and disease. The Bioscience laboratory immune panel regimen included three such tests, and the KU protocol only one.

"some subtle changes in their laboratory parameters." He concluded, nevertheless, that "[c]linically I thought they were normal." The clinical factors which prompted that conclusion were the ages of some of them [seven among the twenty-one were in the sixties and seventies], and the nutritional deficiency of others suggested by red blood indices abnormalities noted in the Midwest/Wheeler tests. The reference range abnormalities notwithstanding, Stechschulte concluded that basically the immune systems were functioning. He deemed it "highly speculative"to suggest "any prognosis as to where their immune systems might be going."[38]

Dr. Stechschulte explained that a disregulated immune system could result in cancer. The latency period to that endpoint is "highly variable," and depends on the strain of the cancer - whether induced by virus, by chemicals or by mutagens. It is known that the use of chemotherapeutic drugs [as in the treatment of the auto-immune diseases] over a period of five to ten years can result in an increased incidence of malignancy in the patient population.

The witness reaffirmed on cross-examination that on the basis of the clinical histories of the plaintiffs, none of them [Mary Landon excepted] suffered a functional abnormality of the immune system. He reaffirmed also that one of the indicia of functional abnormality is the recurrent infection syndrome - such as chronic conjunctivitis, chronic urinary tract infections, chronic rhinitis and chronic pharyngitis. These are conditions, the expert acknowledged, which can be caused by exposure to toxic chemicals. Dr. Stechschulte acknowledged that the histories of the plaintiffs included findings of such chronic symptoms. He

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[38] See APPENDIX K for the evaluation by Dr. Stechschulte of the immune system of individual plaintiffs.

responded nevertheless that these conditions, even if recurrent, were not sufficient to suggest a deficient immune system, unless the symptoms were bacterial in origin.

Dr. Stechschulte explained also that although the medical evidence may find that the plaintiffs suffered from conjunctivitis and other chronic conditions caused by continuous exposure to the toxic chemicals over seven years, that would not necessarily suggest an immune system disregulation [a system out of balance], even accompanied by abnormal laboratory test results. He gave as reasons that conjunctivitis, for example, describes "a group of disorders," some of which may be due to the immune system and others not. Also, he had not come to the diagnosis that these symptoms were recurrent infections. The witness acknowledged that, contrary to his usual clinical procedure, he had not examined any of the plaintiffs - simply because the protocol did not include that procedure.

He acknowledged again that certain toxic chemicals can suppress the immune system. He understood that Alcolac used alkylating chemicals in its production processes. The effects of alkylating agents on the new cell formations, the expert explained, is to alter their DNA and can lead "to killing of those cells." Dr. Stechschulte agreed that toxic chemicals can cause an increase in the IGA antibody production. That is a condition which may be found in the blood cells of a person who suffers malignancy. The KU protocol did not provide for a test of the IGA levels, and none were made under that regimen. The witness agreed that "there were certainly some [plaintiffs] who have elevated IGA levels." Virgil Bradley and Dainie Landon were two among them. Dr. Stechschulte agreed that Bradley and Landon "should be watched and have their IGA levels and other studies done periodically."

He ascribed as the reason, that the IGA "is commonly elevated with chronic inflammation [a]nd chronic inflammation can again be due to a variety of reasons, one of which is malignancy."

The expert witness agreed that it is good medical practice to test every member of a population with symptoms from exposure to toxic chemicals, especially when the exact immunotoxic chemical is not known. He agreed also that it is possible to have an immuno deficiency syndrome even without present disease, but that disease "may be just later down the road." The witness acknowledged that the immune system may change from normal to abnormal, and that such a transition was not subject to prediction.

The cross-examination of immunologist Stechschulte concluded with this exchange:

> Q. Would you agree that these people I represent, who have been impacted by the Alcolac toxic chemicals, should be watched or monitored to determine when such changes might occur?
>
> A. It is appropriate.

## VI

## A
## The Medical Experts and Biological Causation

### For the Plaintiffs

It was the role of Dr. Bertram Carnow to posit that the toxic emissions from Alcolac were the cause in fact of the symptoms, biological manifestations and end points of disease exhibited by each of the plaintiffs. The witness rendered those opinions as a physician, expert in the science of environmental medicine. It is the method of environmental medicine that, to come to a determination as to whether a disease is from an

external cause, the investigator establish by a high degree of probability that the environmental event - here the installation of the Alcolac chemical plant and the subsequent emissions of toxic odors, fumes, foam and explosions - be related to the pathology or disease.

The process of investigation of external cause and diagnosis of disease involves a compound of factors:

> It commences, first, with a related external event - here, the commencement of Alcolac operations and the virtually coincident onset of the emission of fumes, odors and foam discerned by the plaintiffs and other populace in the environs. The related event can be an acute episode - such as an explosion from the monomer reactors, or chronic episodes - emissions over the seven years of Alcolac production.
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> The second factor in the process of investigation of causation and diagnosis of disease is exposure to the event. The demonstration of that element, in the context of the evidence, is the observations of incidents of smoke, vapors and foam and the detection and inhalation of the odors and emissions - followed by symptoms. The evidence was replete with accounts by the plaintiffs and other nonlitigant witnesses, resident in the environs, of observed plumes and incinerations, vapor emissions from Alcolac as well as airborne foam from the bioponds. They described the same composite of odors and assortment of symptoms. The odors, moreover, were characteristic of the toxic compounds involved in the Alcolac processes - the lacquer odor of the methacrylates, the rotten egg sensation of hydrogen sulfide and the sweetness of toluene - among others.
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> The third element is an effect from that exposure. It is an effect which follows the external event and stands in a reasonable temporal relationship with it, whether the exposure is acute or chronic or whether the effect is acute or chronic. An acute effect usually follows soon upon acute exposure. A chronic effect usually follows from repeated exposures over a prolonged time. The acute effects displayed by not only the plaintiffs but also the nonlitigant residents, were on the mucous membranes and the sympathetic nervous system, and manifested also as irritation of the eyes, nose and throat,

headache and vomiting - among the others. The chronic effects from the repeated exposure over the seven year span of toxic emissions were to the major organ systems which were poisoned by the prolonged exposure. It was the conclusion of the environmental medicine expert, for reasons our discussion delineates, that every plaintiff suffered both acute and chronic effects from the acute and chronic exposures to the Alcolac toxic emissions.

The fourth element is whether the effect - here both acute and chronic - is related to the external event, and therefore expected. In the context of the litigation and evidence, the expectedness of the effects of the chemical emissions was demonstrated by the toxicological profiles of each of the toxic compounds prominently involved in the monomer process. The experts drew the toxicological profiles for epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, gylcidyl ether, cyclohexane, methylene chloride, hydrochloric acid, hydrogen sulfide and allyl methacrylate. The experts agreed that many of these toxic compounds induce common physical manifestations, and hence acute effects: epichlorohydrin, allyl methacrylate, allyl alcohol, and ethyl acrylate cause irritation to the eyes, nose, throat, lungs and skin. Exposure to hydrogen sulfide causes dizziness, headaches and vomiting. Chronic exposure to these and the other toxic compounds produces chronic effects to the liver, kidneys and other organs, depresses and destroys the immune system, affects the blood, causes porphyria and even cancer. The chronic effects of two of them is to degenerate the human reproductive system. Epichlorohydrin, ethyl acrylate, toluene, cyclohexane and glycidyl ether all affect the central nervous system. Ethyl acrylate and toluene cause heart arrhythmias or flutters. Cyclohexane causes lethargy, depression, erratic blood pressure and sudden nosebleeds. The evidence was recurrent - as the details of our discussion relate - that the plaintiffs and others exposed to the Alcolac toxic emissions manifested both these expected acute and chronic effects.

The fifth element is to determine if others have been similarly exposed and similarly affected. That component of the causation inquiry, the expert concluded, was readily met by the multiple descriptions of the thirty-one plaintiffs as well as by the sixty nonlitigant witnesses of commonly observed related events - the smoke,

vapors, foam and other emissions from Alcolac, the inhalations of the various odors - common episodes of exposure, common incidences of acute and chronic effects as disclosed by the examinations and tests of the plaintiffs and by descriptions given in testimony by the nonlitigants. It was the remarkable statistics of commonality of exposure and effect among not only the thirty-one plaintiffs, but also among the sixty nonlitigant residents, which heightened the probability to a reasonable medical certainty that the Alcolac emissions were the cause of the diseases diagnosed in the thirty-one plaintiffs. Thus, although the incidence of numbness and tingling in the extremities is 2% in the normal population, among the plaintiff and nonlitigant community, the incidence was between 50% and 60%. That manifestation, as the laboratory tests confirmed, was a symptom of peripheral nervous system abnormality. The laboratory tests of the plaintiffs [as our discussion explains] disclosed that all of the plaintiffs suffer from depressed immune systems and altered cells. They all suffer from abnormal liver metabolism. They all suffer central nervous system damage, except for Lyle Turley, who was not tested because of his youth. The laboratory tests for fertility reported abnormal results for all the seven males who submitted to that procedure. The tests established also that 87% of the plaintiffs suffered some form of porphyria - a very rare disease in the absence of a toxic source. The expert deemed that result "incredible" - and the probability of such incidence in a normal population a quintillion [one billion billion] in the absence of toxic poisoning. It was the unusually high incidence of symptoms, physical manifestations and abnormal laboratory test results of the several bodily organs shared in common by all the plaintiffs which constituted persuasive "markers" for the expert that there was an "extraordinarily strong cause and effect" connection between the offending toxic agents and the manifestations and disease the plaintiffs displayed.

The sixth and final element of the causation inquiry is to determine whether or not the pathology found from the laboratory tests is of the kind expected. The pathology disclosed by the examination of the patients - peripheral neuropathy, memory loss, fatigue, and such manifestations - all was of the kind expected from the exposure to the toxic chemical emission agents. The laboratory results of the immune

> panel studies, the porphyrin counts, and other test procedures [we presently describe] all confirmed injury and damage to the organ systems expected from chronic exposure to the toxic agents involved in the Alcolac production process.

It is evident that to evolve a diagnosis, environmental medicine draws upon other sciences and disciplines - among them, occupational medicine, epidemiology, industrial hygiene and biostatistics. In the case where the point source [the related event] is the operation of a chemical plant, toxicology also impinges. A certification in the environmental and occupational medicine specialty, the expert noted, requires learning and adeptness in each of these cognate disciplines. The expert presented by the plaintiffs to derive causation and diagnosis, Dr. Bertram Carnow, was a polymath whose credentials included board certifications in preventive medicine and the occupational medicine subspecialty, in pulmonary medicine and in toxicology. He was an early founder of the environmental medicine subscience, co-chairman of the national certification board for environmental and occupational medicine and conducted numerous epidemiological studies for government, scientific foundations and labor on the effect of chemical and organic pollutants on human populations.[39] His testimony was received by the court as that of an expert in environmental medicine, occupational medicine, internal medicine, preventive medicine, toxicology and epidemiology.

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[39]The curriculum vitae of the witness, Dr. Bertram Carnow, extends over more than 100 pages of transcript testimony. It draws a portrait of a practitioner, scientist and teacher of international reputation in environmental and occupational medicine. He holds the rank of professor at the Illinois Medical School and at the School of Public Health of the University of Illinois. His advice and expertise has been often engaged by the agencies of the United States and foreign governments as to the effects of toxic chemicals on populations, and other aspects of medical and biological causation.

Dr. Bertram Carnow and Dr. Ruth Conibear [his wife] operate Carnow, Conibear and Associates [CCA], a consultation firm which offers a wide range of occupational and environmental services to government, business and individuals. It maintains a clinic with laboratories, medical equipment and patient facilities. It is supported by a staff of internists, toxicologists, industrial engineers and other professional specialists and consultants.[40] The plaintiffs all presented themselves at CCA in August of 1984 for evaluation over a period of two days by Dr. Carnow. [Four of the plaintiffs - Betty Elam, John Lawrence, Gwendolyn Lawrence and Charlotte Phillips - were previously examined at CCA in September of 1983, the year before.] The patient evaluation rested on three components: symptoms, physical findings and laboratory test results.

It is the postulate of environmental medicine, Dr. Carnow explained, that cause is a cognate of diagnosis, and therefore a cognate of treatment, as well. It is for that critical reason, he said, that to derive a reliable profile that a disease was from an environmental cause, not only must the environmental factors be validly established [that is, Alcolac as the source of the toxic emissions, the quantities, the toxicity of the agents, how they enter the human body, etc.], but also the risk variables of the particular individual afflicted, and the biological end point. The risk variables relate to an assessment of whether the disease diagnosed was altogether the result of chemical poisons or rather of viral origin or of a genetic predisposition. Thus, a genetic deficiency in $alpha_1$ antirypsin predisposes to infections of the lung, as do some toxic chemicals. The risk variables relate

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[40]Fred Boelter, the industrial hygienist who conducted the tests at the Alcolac bioponds in June of 1985 and two previous tests outside the premises, was such a professional staff specialist at CCA.

also to the mode and habits of life of the person. Thus, a cigarette smoker exposed to acrylates, epichlorohydrin or allyl alcohol is more susceptible to severe reaction and disease than a nonsmoker. The biological end point is the effect the toxic chemical has on the body - whether the organs are diseased. In essence, it is another confirmation of causation.

The process of evaluation at CCA began with an interview of each of the plaintiff-patients. It delved intensively into the family, social, occupational and environmental histories of each, as well as the clinical medical history, present illness, symptoms and complaints. The inquiry probed the environmental factors which the patient related to the symptoms and complaints. The response invariably described the sequence of the commencement of Alcolac operations, the incident of odors, vapors, fumes and other emissions, and physical manifestations of irritation to the eyes, nose, throat, skin, and others. The inquiry probed other environmental factors which the patient related to the Alcolac emissions - the appearance of white residue and foam on plant and animal life with the sequel of blighted vegetation and diseased or dead animals. These environmental phenomena, the expert explained, afford insight into toxicity as well as dose, and of the temporal relationship between event and effect so significant to the determination of causation. Another function of the interview was to enable the examiner to determine the laboratory tests to be administered among the thousands available. That determination was aided by an inventory of the bodily systems most predictably affected by exposure to the toxic chemicals used and produced by Alcolac. In that component of the interview each organ system was "gone through to try to help the patient remember things that he might have forgotten."

Dr. Carnow deemed the history derived from the patient interview and the laboratory tests as the most vital aspects of evaluation, and the physical examination less so. The witness explained: physical examination is most informative where the patient presents acute symptoms - such as bleeding, traumatic injury, or other such palpable manifestation. Physical examination is least informative in cases of chronic exposure to toxic chemicals of the kind used at Alcolac because they induce multiple symptoms which mimic other diseases, and so are more difficult to diagnose in the absence of laboratory tests. In such cases, also, physical examination - which for the most part explores the exterior of the body - is least informative to the diagnosis of disease because the toxic chemicals Alcolac emitted are systemic, metabolic poisons which attack and degenerate interior organs. Thus, by the time those manifestations of disease are palpable on physical examination [such as enlargement of the liver, spleen, or other organ], the disease is so advanced that virtually no other aid to diagnosis is needed.

The process of patient evaluation at CCA was conducted under protocols defined by Dr. Carnow and delegated to staff physicians, specialists and consultants. The symptoms gathered from the interview of each plaintiff-patient were classified by a nosologist - a specialist who conforms patient responses into a common symptom according to internationally accepted definition. Dr. Carnow then reviewed the found symptoms and, as validated, sanctioned the entry of the symptom as an abnormality of the SYMPTOMS chart of the plaintiff-patient. The chart was devised to reflect the human organs most susceptible to injury from exposure to the chemicals Alcolac used, produced and emitted.[41] Only those

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[41] See APPENDIX L-1, L-2 and L-3 are typical SYMPTOMS charts as completed and validated by the expert opinion of Dr. Carnow. They reflect the manifestations and complaints of three plaintiff-patients, a random cross-section of the litigants: Dorothy

symptoms validated by Dr. Carnow as caused by exposure to Alcolac toxic emissions or which represented a severe aggravation of a pre-existent condition were noted under the organ affected. The notation was by an orange dot. That determination of symptom rested on the information derived from the 1984 evaluation process at CCA as supplemented by the trial testimony of plaintiff-patient in 1985. The age and life expectancy of each of the plaintiffs, otherwise proven, were superscribed on each chart.

The physical examinations of the plaintiffs then followed and were conducted under the protocol defined by Dr. Carnow and delegated to CCA staff physicians Benjamin and Adamji, board certified internists. The abnormalities the examinations disclosed, again as validated by the expert opinion of Dr. Carnow to be effects of the Alcolac emissions, were entered on the PHYSICAL FINDINGS chart of each plaintiff.[42] The chart was designed to reflect the organ systems most likely to manifest physical findings from exposure to toxic chemicals of the Alcolac ilk. They included the eye, ear, nose, throat and the pulmonary, cardiovascular, gastrointestinal, central nervous, peripheral nervous and endocrine systems. A function of the physical examinations, as well as of the derived symptoms, was to enable an informed selection of laboratory tests among the thousands available for the various organs.

The laboratory tests proceeded under a protocol developed to test those organs and organ functions known to be the biological targets of the chemicals used and emitted by Alcolac:

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Bradley, a female of 74 years; Arnold Sommers, a male of 42 years; and Lance Turley, a male of 22 years.

[42] See APPENDIX M-1, M-2 and M-3 are the PHYSICAL FINDINGS charts as completed and validated by the expert opinion of Dr. Carnow and reflect the physical manifestations of Alcolac damage of litigants Dorothy Bradley, Arnold Sommers and Lance Turley.

epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, glycidyl ether, cyclohexane, methylene chloride, hydrochloric acid, hydrogen sulfide and allyl methacrylate. The number and kind of laboratory tests administered to a plaintiff were governed by the nature of symptoms, physical findings and related diagnostic incidents of the particular evaluation. A LABORATORY chart listed each organ or organ function for which a test was administered, the description of each test, and noted an abnormal result by an orange dot on the chart of that plaintiff. In some instances, as in the case of the male litigants, a fertility analysis test was offered but not taken. Such incidents of nontest were noted on the LABORATORY chart by a gold star.[43] The tests administered ranged in number from 25 for the youths, Amber Cross and Lyle Turley, to 63 for Carl Berry. The tests were conducted at CCA by staff specialists, others by special consultants, and others by special laboratories. The fertility tests were administered at Cook County Hospital, the porphyrin tests were conducted at the Mayo Clinic by the new high pressure liquid chromatography technique, the immune panels done in 1984 were tested by the Bioscience Laboratory in Chicago, and the immune panels ordered in 1985 by Dr. Zahalsky [in consultation with Dr. Carnow] to delve the startling uniformity of abnormalities reported by Bioscience, were done by the Midwest/Wheeler Laboratories in Kansas City. The normal for each test result was usually expressed as a value within the reference range established for the given test.[44] A

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[43] See APPENDIX N-1, N-2 and N-3 are typical LABORATORY charts as completed and validated by the expert opinion of Dr. Carnow. They reflect the abnormal laboratory results of the tests administered to the litigants Dorothy Bradley, Arnold Sommers and Lance Turley.

[44] A reference range used by a laboratory, the expert explained, represents a normal derived from its experience from tests done on masses of population. It expresses the mathematical principle of two standard deviations from the average, itself a reflection of the statistical postulate that test results of random popula-

test value outside the reference range was deemed outside the range of normal, and hence abnormal. An abnormal laboratory result signifies that the body is out of balance - thus [the expert evaluator commented], "it is reasonable to assume that if a

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tions fall along a bell-shaped curve. The postulates of statistics predict that 67% of the test population will fall within the tent of the bell- on both sides of the mathematical mean. That value represents the first standard deviation from the mean. The extension of the curve to encompass 95% of the test population defines the second standard deviation from the mean, and defines normal. The 95% value represents the confidence level required by statisticians that the results of the particular study are not arrived at by pure chance, and hence of statistical significance. See Moultrie v. Martin, 690 F.2d 1078, 1082 (4th Cir. 1982); M. Dore, Law of Toxic Torts § 25.03 [Environmental Law Series, Clark & Boardman, 1987]. The 5% is known in statistics as the p-value. The role of chance is assessed by the p-value, which indicates the probability that an observed association exists by chance alone. A small p-value implies that chance is an unlikely explanation. Occupational Safety and Health Administration, Identification, Classification and Regulation of Potential Occupational Carcinogens, 45 Fed. Reg. 5096 (1980) [OSHA Carcinogen Policy]. The normals of laboratory reference ranges are defined by the average results of 95% of the populations the laboratory has tested. Any test value outside that range is abnormal.

The laboratories which are certified, the expert explained, usually use standards developed by the Bureau of Standards or the Communicable Disease Center. The variations in the reference ranges used from certified laboratory to certified laboratory results from the age and quality of the machines, as well as the test methods used. This is especially true of immunological tests, since the one laboratory may use test antibodies with different characteristics from those used by another laboratory. For instance, the Midwest/Wheeler laboratory used the OKT antibody for various test purposes, whereas the KU laboratory used the Leu antibody for its purposes - each manufactured by a different drug company and each with different characteristics. It was the absence of any reference ranges for the immune panels tested under the KU protocol which prompted both Dr. Carnow and immunologist Zahalsky to the conclusion that the results were meaningless and invalid.

The expert noted that numerous reference ranges, derived from universal statistics, are virtually conclusive: such as blood pressure and pulse rate values. Other normals, as for fertility analysis, are determined by a generally accepted standard.

laboratory test is abnormal, whether high or low, that this is not good." Whether or not the abnormality connotes disease, however, is a matter of medical diagnosis integrated from the history, physical examination and other related data. It was the protocol that an abnormal test result was confirmed by a retest.

The witness reviewed the clinical information amassed from the interview, physical examination and laboratory procedures as to each plaintiff-patient, and rendered opinion on reasonable medical certainty as to those abnormal symptoms, physical findings and laboratory results he deemed to be caused by the Alcolac toxic emissions.[45] That delineation was then evaluated into a diagnosis and prognosis as to each of them. The testimony of Dr. Carnow recurrently noted that the chemicals used by Alcolac - epichlorohydrin, allyl alcohol, and the others - are metabolic poisons to the entire bodily system, and hence required test of all the major organs. He prefaced the evaluations of the abnormal results from these tests and the abnormal symptoms and physical findings manifested by the plaintiff-patients by explanations of the target organ and functions of the Alcolac chemicals, the nature of the tests to detect organ function damage, and the significance of the test results.

> EAR
> A number of the chemicals used and produced by Alcolac are toxic to the 8th cranial nerve, the nerve in the brain which controls hearing. The ear is a high energy system - it works unceasingly - and if the nerves are intoxicated, they damage easily and do not regenerate. People hear at several frequencies, but the frequencies at the upper end are those which first become

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[45]That opinion assumed as true facts the evidence posited by a meticulous hypothetical which recapitulated every detail of the proof presented by the plaintiffs to establish a submissible cause of action. In determination of environmental medical causation, the opinion considered also the mode of life, significance of gender distinctions, the occupational history, genetic disposition and any other factor known to the evaluator which would impinge on the determination of environmental medical causation.

damaged by toxic chemicals. They may also be damaged from severe noise. Thus a person in a very noisy occupation over a period of years may suffer such nerve damage also.

The hearing or audiometric test is conducted in a soundproof chamber and the subject is exposed to noises at different frequencies - at 500, 1000, 2000, 3000, 4000 and 6000 cycles - to determine which frequency the subject can no longer hear. Since those at the upper end - 3000, 4000 and 6000 cycles - are those which become damaged by toxic chemicals, they are the focus of the tests. Abnormal test results at those levels signify nerve damage. A person with a normal ear hears at about 20 decibels; 30 decibels is marginal, and the apprehension of 3000 cycles at 50 decibels signifies a hearing loss. A decibel represents a volume determination at a given frequency. The decibel count is a logarithmic number, so that a change from 40 to 45 decibels at a given frequency, for instance, is not a loss of 5, but almost a doubled loss.

To determine whether in any case the test results represented a hearing loss, the conclusions from the audiometer were applied to the Spoor Graph. That diagram represents a composite calculation of average hearing levels at different frequencies at each age - with a different average number for men and women. The results of the audiometric tests administered to the plaintiffs were applied to the averages on the Spoor Graph to determine the acuity of the hearing of the persons tested. That transposition, toxicology and medical expert Emmett for Alcolac acknowledged, were done accurately.

The test results of twenty among the thirty-one plaintiffs were abnormal. The loss of hearing was attributed to exposure to Alcolac toxic chemicals. Occupation noise or presbycusis [hearing loss incurred by age] were eliminated as a cause in each case.

PULMONARY SYSTEM

The test is the spirometer. The spirometer measures the volume of their intake and the strength of inhalation. It measures FVC - forced vital capacity - the volume of our intake and the strength of exhalation. The subject blows as hard and fast as possible into a large rubber bag. The bag content is measured and compared with a person of the same age, height

and sex. A person with an FVC 80% or more predicted for that age, height and sex is normal. A normal person evacuates 70% to 75% of his air in one second. Any lesser FVC is abnormal and usually attributable to scarring of the lungs. Causes other than toxic chemicals can account for the abnormality. Six of the thirty-one plaintiffs tested abnormally. The abnormalities Dr. Carnow diagnosed were all he deemed caused by exposure to Alcolac emissions.

X-rays were also administered to test the pulmonary system.

CARDIOVASCULAR SYSTEM

The test employed was the electrocardiogram - EKG. It tells about the electric pattern of the heart. The toxic chemicals emitted by Alcolac can irritate the nerve bundle of the heart and damage the heart muscle from want of supply of blood. The pattern described by the EKG informs of any such abnormality.

LIVER METABOLISM FUNCTIONS

The liver is the largest organ in the body and functions as "a big factory." It processes proteins, sugars, fats and fatty substances. The liver regulates porphyrins, iron, enzymes and prothrombin time. It is also the major detoxifying organ in the body. The toxic agents go to the liver to be purified, but because they are so dangerous, they also damage the liver. The liver is the target organ for epichlorohydrin, toluene, ethyl acrylate, glycidyl ether and allyl alcohol - among the chemical agents used and produced by Alcolac. The toxic emissions from the Alcolac processes are metabolic poisons which can affect the numerous liver systems and functions. The laboratory procedures administered were selected to determine which major functions of the liver may have been damaged. They include tests for the metabolism and function of the porphyrins, enzymes, iron, prothrombin time, lipids and proteins.

Porphyrins

The porphyrins are a small factory within the larger factory - the liver. They are combinations of chemicals which function to produce the heme which then mixes with globin to form the hemoglobin of the red blood cells. Hemoglobin transports oxygen from the lungs to the tissues of the body, and so is critical to life. The heme also goes into the production of the hemiprotein P450 enzymes "which run the body." The porphyrins move through the body like a cascade regulated by

a series of dams which supplies them to the body as needed. The heme itself is the end product of original eight porphyrins, broken down through the regulation process.

It is known that epichlorohydrin and some of the other toxic poisons emitted by Alcolac do damage to the system of porphyrin metabolism and the process of regulated flow. Thus, the laboratory test for this liver function was devised to determine any abnormality in the porphyrin system. Until recently, laboratory tests could measure only uroporphyrins and coproporphyrins. A new high pressure liquid chromatography technique [HPLC] available at the Mayo Clinic, however, enables the test and measurement of the entire cascade of porphyrin flow function. Porphyria is the disease which describes the state of abnormal porphyrin metabolism. It has been known as a rare genetic disease, with an incidence of one in an average population of 100,000. It has become known recently that toxic chemicals can also damage the porphyrin system and cause porphyria. Epichlorohydrin can cause porphyria - so can PCB and hexachlorobenzene [the latter two not among the Alcolac chemicals used or made].

Six tests were administered at the Mayo Clinic to the 31 plaintiffs. The tests of four of them disclosed no abnormality. Seven of them exhibited one abnormality, twelve of them two abnormalities, seven of them three abnormalities, and one of them four abnormalities. Thus, the Mayo laboratory results demonstrate that 27 of the 31 plaintiffs [87%] had abnormal porphyrins - some form of porphyria. It was a test finding Dr. Carnow described as "just incredible." The probability of the incidence of porphyria in such a percentage had the Sedalia population not been toxically poisoned - the witness calculated - would be a quintillion [a billion billion]. It was remarkable, as well, he remarked, that 19 of the 31 also had a coproporphia.

Once the system of abnormal porphyrin metabolism "is turned on" the witness explained, "we don't know any way to turn it off." Animal studies show that it might help to reduce the amount of iron in the body - induce anemia. A consequence of the disease may be porphyria crises which are characterized by severe abdominal pain - which some of

the plaintiffs have already experienced. The disease also can affect the central nervous system, cause irritabliity and induce personality change. It can explain, for instance, the sudden impulse to strike a horse between the ears - as did plaintiff Arnold Sommers.

The disease may develop into the stage called porphyria cutanea tarda, where any exposure to the sun causes the skin to blister, but none of the plaintiffs are at that stage.

Enzymes

The liver also produces other enzymes necessary to carry out vital bodily functions, such as proteins to aid in the coagulation of the blood. This function of the liver, along with the others, are the targets of most of the several toxic chemicals identified in the Alcolac production process. There are enzymes that are normally inside a liver cell. When the liver cell has been damaged from chemical intoxication or otherwise, they are produced in abnormal quantity and leak into the blood. GGTP, SGOT, SGPT and alkaline phosphatase are among the enzymes normally within the liver cell. Thus, an abnormally elevated count of these enzymes denotes inflammation of the liver. An increase in the alkaline phosphatase enzyme denotes chronic liver damage - although it may signify increased bone activity, as during the repair of a fracture or in young, growing children. LDH is also an enzyme, but not specific for liver disease. An abnormally high count of LDH also denotes inflammation - although it is not specific for liver disease, but may appear when there is muscle inflammation or alteration of cell function in other organs.

Iron Metabolism

Iron metabolism is usually well regulated, and not many diseases will cause an abnormality of that function. The concern was that since porphyrins use iron in the production of their chemical, abnormal porphyrin metabolism could bring about abnormal iron metabolism. An abnormal test result in iron metabolism, therefore, not only usually reflects abnormal porphyrins, but also an abnormal P450 enzyme system of the liver the porphyrins produce.

Prothrombin Time

The test determines the time it takes to stop bleeding. That function is regulated by vitamin K, manufactured in the liver. A disturbance of vitamin K also disturbs prothrombin time and indicates damage to the liver. The test was repeated because of the unusual number of positive results.

Lipid Metabolism

"Lipid" means "fat" and encompasses an entire group of fatty substances which the liver processes. The lipids tested were HDL cholesterol, LDL cholesterol, cholesterol, triglycerides and lipoprotein phenotype. Abnormal findings as to the metabolism of that group is another mark of liver damage. Since the nerve covers are fatty, abnormality in lipid metabolism can relate to peripheral neuropathy - which is damage to the fatty cover of the nerves. Peripheral neuropathy was a condition found in 15 of the 31 plaintiffs tested.

Protein Metabolism

The liver also regulates protein activity, and so a series of tests were administered to determine whether any toxic chemical damage was done to the protein metabolism function of that organ. The total protein, alpha 2 globulin, gamma globulin and other proteins were tested for metabolism. One test applied was the protein electrophoresis which moves the blood sample in a field and distinguishes among the proteins and counts them. The significance of abnormal test results is that the protein processing function of the liver has been damaged.

GENITOURINARY

A number of the Alcolac toxic chemicals target the kidneys and damage them: epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, glycidyl ether, dimethyl sulfate and hydrogen sulfide. Kidney damage from a toxic agent produces high uric acid, and the tests were devised to determine that fact as well as the state of the kidney function. The tests included urinalysis, BUN [blood urea nitrogen], which determined whether the kidney is getting rid of poisons, as do the creatinine and uric acid tests. Calcium and phosphorus, potassium and chloride counts also indicate the normality or abnormality of kidney functions, and they were also tested.

FERTILITY ANALYSIS

Alcolac chemicals, epichlorohydrin, and allyl alcohol, can cause severe damage to the reproductive organs. It can cause abnormalities of the sperm count in humans and the destruction of testicles in animals. The tests conducted analyze a fresh sperm specimen to determine the count, type of motion, percent of activity, grade and morphology. A single ejaculation produces a normal sperm count of about 100,000,000. A count of between 40 to 60 million is borderline abnormal and a count below 20 million causes difficulty in the fertilization of an egg, especially if they do not move well or remain active for more than four hours. Those represent the conclusions of the recent Wharton study which was the standard Dr. Carnow adopted as authoritative in his evaluations. The tests administered observed the activity of the sperm at intervals: at the end of 30 minutes 80% should continue to move; at 60 minutes, 70%; at the end of four hours, 60% should continue to move. If less than that number is found, it is abnormal because sperm must live for three days to fertilize an egg. The motion of the sperm is also tested. Normal sperm all move in the same direction at once. Random movement in every direction manifests a poor grade of sperm "because that means that they are not going any place." The morphology is observed - whether they appear normal. A 20% abnormal morphology among the 100 million sperm is expected. Most abnormal sperm will not fertilize an egg, and if they do, "they can cause a damaged product."

The fertility analysis was performed on the male plaintiffs amenable to the test. Some of them declined or were not qualified because of prior vasectomy. The seven male plaintiffs tested all displayed at least two abnormal fertility analysis results.

CENTRAL NERVOUS SYSTEM

The central nervous system and brain are targets of many of the toxic chemicals used by Alcolac - among them, epichlorohydrin, allyl alcohol, toluene, gylcidyl ether, cyclohexane, dimethyl sulfate, methylene chloride and allyl methacrylate. The brain is very fatty tissue, so anything that disturbs the lipid metabolism affects the brain. The brain is a high energy and vigorous organ, so anything that depresses the way the body uses oxygen affects the brain functions. It is for this reason that the health of the porphyrin metabolism system, which produces the heme, is important.

Certain brain functions are affected by toxic chemicals, and certain abnormalities manifest - such as attentional deficits, memory loss, depression and others. The tests administered were to quantify any such damage done to the central nervous system. They were administered by Drs. Garron and Wilson, professors of psychology at the Rush Medical Center in Chicago. The tests [30 of them] included the Halstead neurobehavioral battery - which evaluates the visual-spatial function, ability to concentrate and other functions of the brain. The results determine what functions of the brain, how much and where, are abnormal. Each test of the Halstead battery was scored according to a reference range of normals devised for different populations - adults or children, range of education, socio-economic status, and other specific standards. The Beck depression inventory test was also administered. Toxic chemicals which deprive the central nervous system of oxygen induce depression, because depression is the way the brain reacts to loss of oxygen. It is for that reason that depression is a very common result of exposure to toxic chemicals. The Zung anxiety scale test was also administered to determine the anxiety caused by the exposure to the Alcolac toxic chemicals.

PERIPHERAL NERVOUS SYSTEM

The tests of the peripheral nervous system were prompted, among other considerations, by the common symptoms reported among the litigants as well as nonlitigants of numbness and tingling of the limbs and cramps in the legs. The peripheral nervous system, moreover, is the target organ of several of the Alcolac toxic chemicals already repeatedly enumerated. The tests administered were of two kinds: the sensory and reflex tests performed on physical examination and the EMG and PNVC tests performed thereafter by Dr. Subbaru, a CCA staff neurologist. The PNVC tests measured both the motor nerves and the sensory nerves. The measurements are in milliseconds and when a nerve is damaged the nerve impulse slows. The EMG tests the nerve by insertion of thin needles into the muscle the nerve controls at the motor end plate. If there is damage to the nerve at that site, there will be an abnormal EMG result. That signifies axonal degeneration - that the nerve fiber itself, not only the sheath, is dying. That damage is irreversible. The toxic chemicals which Alcolac

emitted are metabolic poisons and cause polyneuropathy - that is, damage to multiple nerves.

The peripheral nervous system of ten of the thirty-one plaintiffs tested abnormally.

MUSCULOSKELETAL SYSTEM

In addition to the EMG, which tested the muscle-nerve relationship, the CPK was administered to determine damage to the muscle. CPK is creatine phosphokinase, an enzyme. "It makes muscle go, and sometimes in toxic conditions, CPK may be abnormally low or abnormally high if there is a specific type of muscle damage."

ENDOCRINE SYSTEM

The toxic chemicals which affect the protein metabolism function of the liver can also affect the thyroid gland of the endocrine system. An abnormality of the P450 enzyme system produced by the porphyrin metabolism of the liver will result in abnormality of the thyroid binding globulin, a protein, which carries the thyroid hormone.

The thyroid gland regulates some of the way in which the body uses oxygen. Two tests were administered to test the thyroid function, a T3 uptake and the T4 RIA. One tests the triidothyronine and the other the thyroxine component of the gland. The thyroid and liver functions are so interrelated that when the T3 tests low and the RIA high, it connotes not an abnormality of the thyroid function, but of protein metabolism - the thyroid binding globulin.

IMMUNE SYSTEM

The chemicals involved in the Alcolac process - epichlorohydrin, toluene and dimethyl sulfate, among others - are toxic to human cells and act to depress the immune system. The condition of chemically induced immune disregulation - the immunologists for the plaintiffs and for Alcolac agreed - may result. The procedures administered by CCA in 1984 and then supplemented by the Midwest/Wheeler laboratory in 1985, were designed to test the T cell and B cell components of the immune system for function and abnormality.[46]

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[46]An abnormal result as to any immune system test, as already related, was noted by an orange dot on the LABORATORY chart of the particular patient-plaintiff. [See n.41, Appendix N-1, N-2 and N-3]. Since the charts were composed by Dr. Carnow at the conclusion of the Bioscience laboratory tests administered by CCA in 1984, they did not provide for any abnormal test result from

The immune system is separated into the B cell and T cell components. The immunoglobulins are that part of the B cell component which helps protect against common infections. The major globulins, G, A and M were tested. An abnormality in the immune system or in protein metabolism may reflect in abnormality of globulins. An abnormal finding in this group means the immunoglobulins are not functioning.

Another series of tests of the immune system was the mitogen challenges. The purpose of these tests was to determine whether the body has adequate defenses - whether the body will attack if it sees an enemy. In a normal immune system, the lymphocytes of both the B cell and T cell components attack potentially harmful invaders. They also attack abnormal cells naturally produced by the body as well as those proliferated by toxic agents. A mitogen challenge is a procedure wherein lymphocytes are taken from the blood and, unstimulated, the energy emitted in counts per minute is tested. That is the unstimulated CPM phase of the mitogen test. Then the unstimulated lymphocytes are presented with an enemy - an antigen - in this case, plant proteins. That should cause the lymphocytes to attack and the counts per minute [CPM] to increase. When the counts increase, that means the lymphocytes are attacking, if they do not increase, that means that they are not attacking. The failure to attack is a test abnormality. The reactions of one type of T cell lymphocyte to the mitogen challenges is reflected in the PHA, CPM and the PHA [phytohemagglutinin] Stimulation Index. The reactions of another type of T cell lymphocyte to the mitogen challenges are reflected in the CON A CPM and CON A CPM Stimulation Index. The B cell lymphocytes were tested by the poke weed mitogen [PWM], another plant protein and the reactions are reflected in the PWM Index. The basic function of the panels administered by Bioscience, therefore, was to test the immune functions of the cells - that is, "when they see an enemy, do they attack the enemy?" It was the

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the Midwest/Wheeler tests. The orange dots for those abnormal results were entered at the direction of Dr. Carnow by counsel at the trial and appear on each LABORATORY chart. Midwest/Wheeler tested not only the white blood cells, however, but also the red blood cell system, and any abnormality in the latter system is noted by an orange dot aside the HEMA legend. The nature of the abnormality of either Midwest/Wheeler test was explained in the course of the Carnow testimony.

unusual findings of T cell abnormalities from these mitogen challenges that prompted Dr. Carnow to confer with immunologist Zahalsky and then to arrange for further tests of the patient-plaintiffs by the Midwest/Wheeler laboratory.

The supplemental procedure conducted by Midwest/Wheeler in 1985 not only further tested the immune system but also the hematologic [blood] system.

Other immune system tests administered by CCA and conducted by the Bioscience laboratory in 1984 were to the white cell populations of the patient-clients. They are part of the defense system, and the segs subpopulation increases when infection is present. The tests also counted the total lymphocytes, and eosinophil, monocyte and basophil populations.

The basic function of the panels administered by Midwest/Wheeler was to further define the subpopulations of the lymphocytes, the T cells and the B cells. The purpose of the tests was to determine the kind of T cells in the lymphocyte populations - the helpers, suppressors, killers or memory T cells, and also whether the relationships are out of balance. These test results inform whether or not there may be suppression in the bone marrow, or whether or not the T cells are unprogrammed when they leave the lymphatic tissue - and other such insights.

It was the conclusion and opinion of Dr. Carnow that the Midwest/Wheeler tests determined that the patient-plaintiffs exhibited immune system abnormalities in multiple areas, "particularly of the T lymphocyte series," that the T lymphocytes are "one of the major defenses against cancer," and that it is known that the T lymphocytes can be damaged by toxic chemicals of the kind used by Alcolac. It was the conclusion of the expert, also, that the Midwest/Wheeler results demonstrate and confirm that "the immune systems of most of these Sedalia people were very severely depressed because of damaged organs" from exposure to the Alcolac emissions

The purpose, role and function of each of the laboratory procedures thus elaborated, Dr. Carnow then evaluated the symptoms, physical findings and abnormal laboratory results as to each

of the patient-plaintiffs [within the context of a detailed personal, clinical and occupational history of each as augmented by the trial testimony] to derive a diagnosis of the biological pathology and disease caused to each organ or function by the Alcolac toxic chemical emissions.[47] The symptoms, physical findings and abnormal laboratory results found - as well as the diagnosis as to each plaintiff-patient were those evaluated as attributed to the Alcolac operations by a reasonable medical certainty.[48]

ETHEL BERRY AGE 73 LIFE EXPECTANCY 13.78 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including

1) recurrent conjunctivitis
2) bilateral hearing loss with probable neurosensory 8th cranial nerve damage
3) chronic rhinitis and pharyngitis
4) chronic chemical bronchitis
5) arteriosclerotic heart disease with angina, chronic insufficiency and hypertension
6) liver dysfunction including
   a. chemical hepatitis
   b. abnormal lipid metabolism
   c. porphyria
   d. abnormal protein metabolism
7) chronic obstructive uropathy with hypercalcemia

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[47]See: APPENDIX O-1, O-2 and O-3 are the DIAGNOSIS charts as completed and validated by the expert medical opinion of Dr. Carnow and represent the biological damage caused by the Alcolac toxic chemicals to litigants Dorothy Bradley, Arnold Sommers and Lance Turley. The witness completed and validated a like DIAGNOSIS chart for each of the patient-plaintiff litigants.

[48]The symptoms, abnormal findings, and abnormal laboratory test results Dr. Carnow attributed to exposure to the Alcolac toxic agents [and from which diagnosis derived] were entered as to each plaintiff as a dot according to the bodily organ affected. Thus, three summaries were composed and received as demonstrative exhibits: SUMMARY OF SYMPTOMS, SUMMARY OF ABNORMAL PHYSICAL FINDINGS, SUMMARY OF ABNORMAL LABORATORY TESTS. They appear as APPENDIX P-1, P-2, P-3. The large dot in each summary indicates that the organ under which it was entered was affected by the Alcolac toxic agents. The smaller dot[s] entered alongside indicate[s] the number of abnormal symptoms, finding, or laboratory test result as to that organ.

8) depression
9) peripheral neuropathy
10) immune system dysfunction with suppression
11) recurrent chemical contact dermatitis
12) hyperglycemia with early diabetes

PROGNOSIS: Very guarded to poor.

The diagnosis entered by Dr. Carnow as to Ethel Berry - Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems - was that determined for each of the thirty-one litigants, although by distinctive manifestations. The witness then explained - in terms of the symptoms, physical findings, laboratory results and environmental history of the litigant - how, as a medical exercise, the subelements of diagnosis were derived:

1) Recurrent conjunctivitis rests on symptoms and history of acute problems with the eyes which recurred every time she was exposed to the Alcolac chemical emissions: redness of the eyes, burning, itching. Although the evaluator considered it more probable that there was residual damage to the eye and so the diagnosis was likely chronic conjunctivitis, the residual damage to the eye was not palpable, therefore, recurrent and not chronic conjunctivitis was attributed.

2) Bilateral hearing loss with probable neurosensory 8th cranial nerve damage rests on the abnormal audiometric tests at the upper frequencies, a sign of bilateral nerve damage to the 8th cranial nerve by the Alcolac chemicals.

3) Chronic rhinitis and pharyngitis rests on physical findings of swollen nose with exudate material within. Rhinitis describes the chronic inflammation of the mucous membrane of the nose. It was a chronic, rather than a recurrent condition, because the swelling and residue were evident even when there was no exposure.

4) Chronic chemical bronchitis rests on findings that the bronchial tubes were chronically inflamed. In response to cross-examination, the expert acknowledged the work history of Ethel Berry - that she worked for 23 years in a broom factory, a very dusty occupation and developed a low grade abnormality from that exposure. It was an abnormality different from the bronchitis and is in the nature of chronic restrictive pulmonary disease, rather than bronchial disease. The restrictive pulmonary disease caused by the dust involves the lung tissue itself. The chronic chemical bronchitis involves the bronchial tubes irritated by the toxic chemicals. The bronchitis was diagnosed from the inflamed bronchia from recurrent episodes of inhalations of obnoxious Alcolac odors, which sometimes burn and irritate and induce nausea. The odors also induce productive coughs. The diagnosis was limited to bronchitis and did not include the restrictive pulmonary disease to the lung tissue because it was attributable to occupation, and not to the Alcolac chemicals.

5) Arteriosclerotic heart disease with angina, chronic insufficiency and hypertension rests on findings of pain on exertion from chronic want of sufficient blood supply to the heart, with the consequence of recently developed high blood pressure. It included the diagnosis that the blood vessels of the heart have been adversely affected by the abnormal fats in the blood developed from the intoxication of the liver. The diagnosis took into account her prior medical episode of hypertension, since for several years Ethel Berry has been free from any such disease. It also took into account that several of her brothers died of heart disease, but discounted that information since heart disease of that kind is not of genetic significance.

6) Liver dysfunction manifested by chemical hepatitis, abnormal lipid metabolism, porphyria and abnormal protein metabolism rests on the abnormal laboratory test results as to

porphyrins, enzymes and lipids. The tests for the enzymes GGTP, SGOT and SGPT show abnormal elevations, indications that the liver is acutely inflamed - a condition of hepatitis attributed to the toxic chemicals. In addition the alkaline phosphatase was also abnormal. The lipid metabolism function of the liver was also diseased. The cholesterol was abnormally high at 291, and in addition the lipoprotein phentotype was abnormal and the triglycerides were extremely high at the 407 level [normal, 150]. The inflammation of the liver and the impairment of the liver function is due to the destruction of cells - a direct action of such toxic chemicals as epichlorohydrin, and the intoxication of the systems of liver metabolism by others. The very abnormal fats in her blood demonstrated by the lipid metabolism tests show very abnormal fats in the blood, developed from the chemical intoxication of the liver. The porphryn metabolism was also diseased. The tests show abnormal heptacarboxylic porphyrins and an abnormal coproporphyrin ratio. The abnormal porphyrins means that the porphyrin factory and the enzymes which depend on the heme production of the porphyrins are not functioning normally. The abnormal protein metabolism means that the body tissues, the end product of the protein metabolism, are not being properly produced.

7) Chronic obstructive uropathy with hypercalcemia rests on the test result of abnormal levels of calcium in her blood which inflames the genitourinary system and renders urination difficult. It represents an abnormal function of the kidney in this case.

8) Depression rests on not only the Beck depression inventory test, but also on the symptoms, findings and history. It represents a mild abnormality, but nonetheless attributed to the Alcolac chemical activity. There may be some psychogenic overlay but that is the kind of depression manifested by people exposed to toxic agents of the kind emitted. She had some difficulty with

some other tests - such as the visual-spatial perception, but the clinical significance of that result was discounted. The expert acknowledged to the cross-examiner that her condition of poor circulation to the brain as well as domestic stress may have combined with the toxic agents to cause the depression. He acknowledged that the Beck inventory was not as definitive as a CAT scan or other such procedures.

9) Peripheral neuropathy rests on the absence of ankle reflex, decreased sensation to pinpricks in sites of the nerves on the left hand, as well as the symptoms of tingling limbs, pains, cramps and physical findings of swelling in the legs.

10) Immune system dysfunction with suppression rests on the "whole host of abnormalities" the 1984 Bioscience and the 1985 Midwest/Wheeler immune panel results disclose. These test abnormalities show that "[h]er whole T cell immune system is depressed," and a "severe depression of her [immune] system."

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | slightly abnormal gamma M at 79 [normal, 80 to 310] |
| Mitogen Challenge: | abnormal PHA stimulation net CPM at 41,800 [normal, over 62,000] |
| | abnormal PHA stimulation index at 92 [normal, over 130] |
| | abnormal CON A stimulation net CPM at 10,334 [normal, over 12,000] |
| | abnormal CON A stimulation index at 24 [normal, over 40] |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormal elevation of mature B cells at 629 [normal, 50 to 570] |
| | abnormal B cell percentage at 30.3% [normal, 5 to 15%] |
| | abnormal depression of natural killer T cells at 5.4 [normal 8 to 22] |
| Hematologic system: | abnormal ring of distribution [width] of red blood cells |
| | abnormal increase in mean platelet volume |

abnormal size of red blood cells
[anisocytosis]
abnormal lack of red color
[hypochromia]

11) Recurrent chemical contact dermatitis rests on symptoms of burning of the skin and abnormal pigmentation of the skin related to the incidents of exposure to the Alcolac emissions.

12) Hyperglycemia with early diabetes rests on the test result of increased sugar in the blood. Her prior medical history and records indicate a normal level of blood sugar and that she was free of the stigmata of diabetes. A condition of diabetes could account for some elevation of the lipids, but no such condition was suggested from her history.

PROGNOSIS: Very guarded to poor.

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The evaluations of the symptoms, physical findings and laboratory results of the other patient plaintiffs, as well as the diagnoses and prognoses derived from them were rendered by Dr. Carnow, in turn. The diagnosis and prognosis of each is rescripted, as well as the summary of the symptoms, physical findings, laboratory results and other data on which they rest.

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CARL C. BERRY AGE 54 LIFE EXPECTANCY 25.51 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) bilateral chronic conjunctivitis
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic rhinitis and pharyngitis
4) liver dysfunction including
   a. abnormal lipid metabolism
   b. abnormal porphyrins with coproporphyinuria and abnormal porpobilinogen
5) reproductive system damage
6) cardiac dysfunction with:

a. tachycardia
b. chest pain
c. shortness of breath

7) central nervous system dysfunction with depression
8) severe immune system dysfunction with suppression
9) abnormal muscle metabolism with abnormal CPK

PROGNOSIS: Very guarded to poor.

The witness then explained the data on which the sub-elements of diagnosis rest:

1) Bilateral chronic conjunctivitis rests on the symptoms of burned and watery eyes with redness related to the exposure of the vapors, smoke and fumes from Alcolac, as well as on the inflammation of the eyes found on physical examination. These manifestations followed the presence of odors with the smell of lacquer [which the witness identified as the methacrylates] as well as the vapors, smoke and fumes from Alcolac. The conjunctivae are the delicate membranes which cover the eyes. The witness distinguished between recurrent conjunctivitis and chronic conjunctivitis - one condition or the other manifested by every patient-plaintiff. The condition is recurrent when red eyes result upon the repeated exposures but are not present at the time of physical examination. The condition is chronic when the eyes exhibit inflammation upon examination even absent recent exposure, or when there is a history of redness of the eyes "all the time," or when the eyes are matted in the morning, or gritty.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on the abnormal results from the audiometric tests. They show bilateral neurosensory hearing loss from the 3000 to the 6000 frequency ranges. Normal hearing apprehends speech between the 500 and 3000 frequency ranges, and sound up to the 6000 frequency. Those toxic chemicals which attack the central nervous system characteristically first cause damage to the 8th cranial nerve at the upper ranges. It was for that reason that the tests were for the 3000 to 6000 frequency ranges. The

tests administered to Carl Berry at those ranges gave abnormal results throughout. At the 3000 frequency range, for instance, normal hearing apprehends sound at no higher than 25 decibels. Carl Berry could hear that sound only at 50 decibels. Hearing loss was also evident at 4000 and 6000 cycles. The results demonstrated damage to both 8th cranial nerves - a bilateral neurosensory hearing loss from the Alcolac toxic chemicals.

It is damage which does not regenerate, and is irreversible. Hearing loss at those frequency levels may also be the result of occupational noise. Carl Berry was a heavy equipment operator, but never experienced any hearing loss from the practice of that occupation. He was also a signal officer on an aircraft carrier, but he wore ear covers which excluded the noise. His tests for hearing were satisfactory before release from the service. The witness acknowledged also that presbycusis - hearing loss due to age - is a condition that may affect hearing at all frequencies. The loss of hearing due to presbycusis, however, does not "notch," but increases as the frequency increases. Hearing losses due to chemicals or noise, on the other hand, do "notch." That was demonstrated in the tests administered to Carl Berry which show that he heard at the 4000 range at 50 decibels and at the 6000 range at 45 decibels - a slight improvement at the higher frequency. The full history and other clinical data led the expert to the conclusion that the hearing loss was not from occupational noise, but from the intoxication caused by the Alcolac emissions. It also excluded presbycusis as a relevant factor in the subdiagnosis of the manifested bilateral hearing loss both on the pattern of "notched" hearing loss, uncharacteristic to presbycusis, but also on the basis of studies which attribute the disease to noisy industrial environments. Carl Berry, rather, lived in the rural environs of Sedalia.

3) Chronic rhinitis and pharyngitis rests on symptoms of irritation to the nose and soreness to the throat from the Alcolac odors and emissions. There was also the physical finding of mucous membrane irritation of the nose. The irritation of the mucous membrane, when chronic, impairs the production of tears, and also renders the person more susceptible to infection. In terms of diagnosis, the irritations are markers of a direct relationship between the discerned vapors, smoke and smells from Alcolac and the manifested symptoms and conditions.

4) Liver dysfunction including abnormal lipid metabolism, abnormal porphyrins with coproporphyrinuria and abnormal porphobilinogen rests on the normal results of the laboratory tests. The abnormal lipid metabolism dysfunction of the liver was shown by the result of 28 in the HDL cholesterol test with a normal reference range of 29 to 77. The studies show, the witness explained, that although the reference range extends from 29 to 77, a test value below 45 means that the person is "at increasing risk" - therefore, a person at 29 is "at high risk" of cardiac disease. The reference range used by Smith-Kline, the laboratory which conducted the HDL cholesterol test, is based on the Framingham epidemiologic study which concludes that an HDL value of 45 in a male and below 55 in a female means the person is "at increasing risk" of heart disease. The test result for Carl Berry demonstrated abnormal metabolism of the fats. The expert acknowledged that the discontinuation of smoking will improve the HDL count, but Berry had not smoked since 1969, and so that earlier life habit was not significant to the causation of the manifestation. The porphyrin metabolism function of the liver also tested abnormally. It demonstrated a condition of porphyria, coproporpyrinuria and porphobilinogenuria.

5) Reproductive system damage rests on the abnormal results of the fertility analysis tests. The normal range of count for fertility is between 60 to 200 million sperm per specimen. That represents the conclusions of the recent Wharton study which was the standard Dr. Carnow adopted as authoritative in his evaluations. [The witness, himself, conducted some work for the World Health Organization on the subject.] The sperm count Carl Berry produced was above the 60,000,000 level, but there were abnormalities in the morphology and motility of the sperm. The witness explained that there was "good evidence, from the animal data, that there is suppression of sperm production and destruction of testicular tissue by epichlorohydrin and allyl alcohol," among the compounds used in the Alcolac operation.

6) Cardiac dysfunction with tachycardia, chest pain and shortness of breath rests on symptoms and physical findings of abnormal pulse, pain in the chest and, particularly pain in the chest upon walking fast or exertion, and shortness of breath. Those together constitute a syndrome of dysfunction. They suggest that the blood supply to the heart is not sufficient. They suggest a heart which does not compensate well, and a coronary insufficiency.

7) Central nervous system dysfunction with depression rests on the abnormal result of the Beck inventory test of 19 [normal, 0 to 10], as well as on the abnormal digital vigilance test result.

8) Severe immune system dysfunction with depression rests on the abnormal results from both the 1984 Bioscience and the 1985 Midwest/Wheeler tests.

[Bioscience Laboratory Test Results, 1984]

Immune system:

| | |
|---|---|
| Immunoglobulin: | abnormal gamma M at 50 [normal, 80 to 310] |
| Mitogen challenge: | abnormally decreased PHA stimulation net CPM |
| | abnormally decreased CON A stimulation net CPM |
| | abnormally decreased CON A stimulation index |

[Midwest/Wheeler Laboratory Test Results, 1985]

Immune system:

| | |
|---|---|
| Lymphocytes: | abnormal OKT-10 cells at 32.9 [normal, 0 to 15] [OKT-10 are immature, unprogrammed lymphocytes, probably preleukemic cells] |
| | abnormally elevated NKH-1 [natural killer] cells at 19.7 [normal, 3.8 to 9.6] |
| Hematologic system: | abnormally shaped red blood cells [poililocytosis] |
| | abnormal sizes [anisocytosis] |
| | abnormal color [hypochromia] |

9) Abnormal muscle metabolism with abnormal CPK rests on the symptoms of soreness and tingling in the left elbow which weakens the grip on objects and the abnormal CPK test result of the related musculoskeletal system. The symptoms signify both sensory and motor abnormalities of the ulnar nerve. That nerve comes down from the shoulder into the elbow, and the ingestion of metabolic poisons may cause it to swell. That results in pain and loss of

the muscular function of the arm. CPK [creatine phosphokinase] is the enzyme which appears in abnormal quantity when there is muscle injury. The test for CPK yielded an abnormal result, which signifies an abnormal muscle metabolism from exposure to the Alcolac chemicals.

JACQUELINE BERRY AGE 55 LIFE EXPECTANCY 28.61 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) recurrent conjunctivitis
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic rhinitis and pharyngitis
4) liver dysfunction including:
   a. abnormal LDH - possible chemical hepatitis
   b. abnormal porphyrin ratio with borderline coproporphyrinuria
   c. abnormal protein metabolism
5) central nervous system dysfunction including
   a. brain dysfunction
   b. depression
6) peripheral polyneuropathy
7) abnormal muscle metabolism
8) recurrent contact dermatitis with probable hypersensitivity state
9) immune system dysfunction with suppression
10) abnormal electrolytes with probable renal dysfunction

The witness then explained the data on which the subelements of diagnosis rest:

1) Recurrent conjunctivitis rests on symptoms and history of irritation, redness and watering of the eyes with blurred vision from exposure to the odors, smoke, haze and other emissions from Alcolac.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on abnormal audiometry results of the upper frequencies with borderline abnormalities in the lower frequencies. It is a characteristic phenomenon that damage to the

8th cranial nerve from toxic chemicals starts at the high frequencies and spreads to the lower frequencies. The hearing loss to Jacqueline Berry was to both 8th cranial nerves, a bilateral neurosensory hearing loss.

3) Chronic rhinitis and pharyngitis rests on the irritation to the nose and burning in the throat from the Alcolac odors and emissions. Chronic rhinitis rests also on the physical finding of swollen mucous membrane.

4) Liver dysfunction including abnormal LDH [possible chemical hepatitis], abnormal porphyrin ratio with borderline coproporphyrinuria and abnormal protein metabolism rests on the abnormal results of the laboratory tests of the protein and porphyrin metabolism functions of the liver. The LDH enzyme tested at 141, elevated from the 62 to 131 normal. That represents a possible inflammation of the liver, and hence a possible hepatitis. In addition, another liver function, the coproporphyrin to uroporphyrin ratio, usually 2 to 1 to 6 to 1, was elevated to 7 to 1. Although the other enzymes - the GGTP, SGOT and SGPT - were normal, the other abnormalities found in the liver, the abnormal protein metabolism, as well as the known action of these toxic agents suggest that the abnormal LDH may represent tissue damage as well as alteration of the tissue function. That is why the evaluator termed the manifestation a "possible chemical hepatitis."

The abnormal protein metabolism was manifested by a T3 uptake deficiency, which, although a part of the endocrine system, did not represent a thyroid deficiency, but an abnormality of the thyroid binding globulin - a protein manufactured in the liver.

The abnormality of the coproporphyrins was termed "borderline" because her coproporphyrins were at 56 on a scale of 3 to 56. Although, strictly speaking, the count was within the

normal range, since it rests precisely at the two standard deviations and about 25 from the mean, the evaluator described the finding as "borderline." That evaluation was reinforced, moreover, by the phenomenon of the multiple incidences of porphyrin abnormality among those tested, as well as by the known ability of these Alcolac chemicals to cause abnormal porphyrins. The witness was confident in the integrity of the test results as a basis for subdiagnosis because the high power liquid chromatography process used by the Mayo Clinic for that purpose not only measures fractionates and the porphyrins precisely, but also conducts the test on a reliable sample of urine collected during the bodily 24 hour diurnal cycle, and then repeats abnormals.

5) Central nervous system dysfunction including brain dysfunction and depression rests on a neurobehavioral battery abnormality as well as on an abnormal Beck depression inventory test, and physical findings of abnormality to the 7th and 8th cranial nerves. The history taken by the CCA examiner, Dr. Garron, notes that Jacqueline Berry used Indural, a drug which on rare occasions causes some depression. It was a datum not clinically significant, however, since the tests administered by Dr. Garron resulted in several central nervous system abnormalities: the digital learning test, the digital vigilance test, as well as other areas. That represents diffuse brain damage. There was also damage to the cerebellum, the back brain which controls balance and other critical functions. It was the opinion of the evaluator that, given the nature of the toxic chemicals to which she was exposed, as well as the found abnormalities of the 7th and 8th cranial nerves and the neurobehavioral test results, the depression was part of her syndrome.

6) Peripheral neuropathy rests on symptoms of sensory abnormalities - muscle cramps, numbness in the hands, aches in the arms, and charley horses in the calves of her legs. The EMG

[electromyograph] test result [a nerve conduction velocity test] was normal. The witness explained that it would be expected that a person with neuropathy would manifest abnormal reflexes, abnormal pin stick, abnormal EMG and abnormal symptoms. That, however, is usual when the pathology is far advanced. The normal EMG does not rule out abnormalities, but shows only that the EMG test gave a normal result. Multiple tests are done and multiple parameters considered. In this case, the subdiagnosis rests on the symptoms of peripheral neuropathy.

7) Abnormal muscle metabolism rests on symptoms and findings of abnormal muscle function as well as on the test which shows the presence of the CPK [creatine phophokinase], the enzyme which shows abnormally when there is pervasive muscle injury.

8) Recurrent contact dermatitis with probable hypersensitivity rests on symptoms of itching, skin rashes and what is known of the Alcolac toxic chemicals to which the patient plaintiff was exposed. The acrylates and methacrylates not only cause severe skin damage on contact, but they also sensitize the skin even in the absence of actual contact. Contact dermatitis relates to the severity of the toxicity of the given chemical according to the degree of contact. When the skin has become hypersensitive a small exposure can cause severe reactions.

9) Immune system dysfunction with suppression rests on the abnormal tests results administered by Bioscience in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Mitogen challenge: | abnormal PHA stimulation net CPM at 44,288 [normal, over 62,000] |
| | abnormal PHA stimulation index at 118 [normal, over 130] |
| White blood cells: | abnormally depressed total count at 4100 [normal, 4500 to 11,000] |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormal OKT-4 [helper] cells |

| | |
|---|---|
| | abnormal depression of NKH-1 [natural killer] cells |
| Hematologic System: | abnormally increased mean corpuscular hemoglobin |
| | abnormally increased random distribution width |
| | abnormally increased mean platelet volume |

10) Abnormal electrolytes with probable renal dysfunction rests on the abnormal genitourinary test results. The electrolytes - the potassium, sodium and chlorides - were abnormal and signified kidney damage. It is the function of the kidney to control the electrolytes, elements critical to life, in a delicate balance. If the electrolytes are too high or too low, the body function becomes imperilled. Some medications - such as high blood pressure pills - can depress the potassium. In her case, however, the potassium as well as the chlorides are abnormally high. These abnormalities represent the abnormal handling of these electrolytes by the kidney, induced by the attack made on that organ by a number of the toxic chemicals generated and emitted by Alcolac.

PROGNOSIS: Very guarded.

VIRGIL BRADLEY AGE 75 LIFE EXPECTANCY 10.67 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) bilateral neurosensory hearing loss with 8th cranial nerve damage
2) chronic conjunctivitis
3) chronic rhinitis and pharyngitis
4) chronic chemical bronchitis - in remission
5) liver dysfunction including:
   a. abnormal lipid metabolism
   b. abnormal iron metabolism
   c. abnormal porphyrin metabolism
6) reproductive system dysfunction
7) diffuse brain damage with dementia
8) peripheral polyneuropathy
9) polyarthralgia and polymyalgia
10) hyperglycemia - rule out diabetes
11) immune system dysfunction
12) renal insufficiency with mild uremia

13) hypertension
14) recurrent chemical dermatitis with probable hypersensitivity component

The witness then explained the data on which these components of subdiagnosis rest.

1) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on the abnormal audiometric test results throughout the 500 to 6000 frequency ranges - very severe at the higher frequencies and less so at the low frequencies. This represents a course of nerve damage, still spreading. The hearing loss is bilateral and extensive: a 95 decibel loss at the 6000 cycle, a 70 to 75% at 4000 cycle, a 65% loss at the 3000 cycle, and a 30 to 35% loss at the 500 cycle.

2) Chronic conjunctivitis rests on symptoms of irritation, redness and watering of the eyes following exposures to the Alcolac odors and emissions. The inflammation of the eyes remains palpable even without exposure.

3) Chronic rhinitis and pharyngitis rest on the symptoms and findings of an irritated and swollen mucous membrane, sore and swollen throat. These conditions followed exposures to the Alcolac toxic odors and emissions and continued absent exposure.

4) Chronic chemical bronchitis - in remission rests on symptoms and extensive history of severe coughs following exposure to the Alcolac chemical emissions. The symptoms were in remission at the time of physical examination. The coughs were productive of mucus. The witness discounted that the former occupation of the patient plaintiff as a meatpacker bore on that element of diagnosis.

5) Liver dysfunction including abnormal lipid metabolism, abnormal iron metabolism, abnormal porphyrin metabolism rests on the abnormal results of the tests for those functions of

the liver. The test of the lipid metabolism function shows triglycerides of 155 with a reference range of 30 to 150. The uroporphyrins of the porphyrin metabolism are at 4 with a reference range of 8 to 44. The iron metabolism was abnormally low. The low serum iron suggests some disturbance of the iron-porphyrin cycle. The condition of low uroporphyrins is not as serious as if it were high, but it signifies an abnormality in porphyrin metabolism nonetheless.

6) Reproductive system dysfunction rests on the abnormal results of the fertility analysis. Virgil Bradley was then 75 years of age, but although age is a significant factor as to impotency, it is not as to fertility. The results show a range of abnormalities: the sperm count was abnormally low at 36 million [normal, over 60 million], contained 5% of primitives - a pathological abnormality - only 25% of the sperm were alive after 30 minutes [rather than the normal 80%] and only 15% were alive after 4 hours [rather than the normal 60%].

7) Diffuse brain damage with dementia rests on the abnormal results of the neurobehavioral battery. Dementia in the context of the subdiagnosis means diffuse brain damage. It represents a "very significant loss of thinking power" - a condition "more serious than diffuse dysfunction."

8) Peripheral polyneuropathy rests on symptoms of sensory abnormalities, physical findings of absence of deep tendon reflexes and an abnormal result on the PNVC [nerve conduction] test to the upper left extremity.

9) Polyarthralgia and polymyalgia rests on acute and chronic symptoms of joint stiffness or crepitance, joint aches and muscular pains and cramps following exposure to the Alcolac emissions. Arthralgia describes a condition of pain in the joints. Arthritis describes inflammation or swelling in the joints. The subdiagnosis was valid, the expert witness Carnow explained,

because there was no evidence of degenerative joint disease, nor of deformity, nor of osteoarthritis. He discounted a medical record of Virgil Bradley, dated March 26, 1982 - from some unidentified source which diagnoses degenerative arthritis - since there was no arthritis present at the time of the examination at CCA.

10) Hyperglycemia - rule out diabetes rests on an abnormal glucose in the endocrine system. The result of that test was 141, with a normal reference range of 70 to 110. The medical history discloses a glucose elevation of 192, with a normal reference range of 80 to 120 used by that laboratory, for that test conducted in 1971. That was the only incidence of abnormal blood sugar in the previous medical record, however, and since there was no indication that it was a fasting blood test, Dr. Carnow could not attribute any significance to that record of abnormality. If that represented a condition of diabetes, the witness observed, Virgil Bradley would be "in very serious trouble by now without any treatment." The test was never repeated, so the witness concluded it was specious. The abnormally-elevated glucose, the witness determined, demonstrated not diabetes, but suggested a pancreatic abnormality or deficiency.

11) Immune system dysfunction rests on the abnormal results of the Bioscience tests in 1984 and of the Midwest/-Wheeler tests in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormal globulin A |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormal elevation in OKT-8 [suppressor cells] |
| | abnormal elevation in OKT-10 cells |
| | abnormally depressed T-4/T-8 ratio [an excess of suppressor to helper cells] |
| | abnormally elevated B-1 [mature B] cells at 18.4 [normal, 5 to 15] |
| | abnormally elevated natural killer |

cells at 32 [normal, 8 to 22]
abnormally elevated NKH-1 [killer suppressor] cells at 15.3 [normal, 3.8 to 9.6]

Hematologic system: abnormally elevated mean corpuscular volume
abnormally elevated mean corpuscular hemoglobin
abnormally elevated random distribution
abnormal sizes [anisocytosis]

The witness concluded that the test results of six immune system abnormalities in 1985 contrasted to one such abnormality in 1984 suggests a deterioration of the Virgil Bradley immune system. The 1985 test results from Midwest/Wheeler represent "gross abnormalities of the kinds of lymphocytes and the ratios of lymphocytes in the blood" and shows "a profound disturbance of the system." The mitogen challenge tests which yielded no abnormal result in the 1984 Bioscience tests, however, were not repeated, therefore the current state of that element of the immune system is not known.

12) Renal insufficiency with mild uremia rests on the abnormal results of the creatinine and uric acid tests of the genitourinary system, as well as on symptoms of nocturia and difficulty in urination. The creatinine and uric acid tests were both abnormally elevated. An elevation in creatinine is a sign of early kidney failure. The renal insufficiency subdiagnosis means that the kidneys are not getting rid of the poisons as they should. The mild uremia signifies that, although not great at present, the abnormality means a "buildup of poisons in the body."

13) Hypertension rests on the physical examination at CCA. The prior medical history confirmed to the witness that the hypertension was a condition which emerged in the 1980s.

14) Recurrent chemical dermatitis with probable hypertensivity component rests on symptoms of skin rash, itching and burning and skin lesions consequent upon the inhalation of odors and other Alcolac emissions.

PROGNOSIS: POOR.

DOROTHY BRADLEY AGE 74 LIFE EXPECTANCY 13.05 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) conjunctival damage with hemorrhage
2) bilateral hearing loss with 8th cranial nerve damage
3) chronic rhinitis
4) liver dysfunction including:
   a. abnormal LDH
   b. abnormal iron metabolism
   c. abnormal porphyrin metabolism
5) early renal insufficiency
6) central nervous system dysfunction including:
   a. cerebellar damage
   b. diffuse cerebral damage
7) peripheral nervous system dysfunction
8) immune system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Conjunctival damage with hemorrhage rests on symptoms of irritation of the eyes and soreness in the eyeballs following the exposure to and inhalation of the odors and other Alcolac emissions. That subdiagnosis rests also on the findings at the time of physical examination.

2) Bilateral hearing loss with 8th cranial nerve damage rests on the symptom of loss of hearing acuity as well as on the audiometric test results of loss of hearing throughout the 500 to 6000 range of frequency cycles. The abnormal results represent a neurosensory hearing loss, most severly at the high frequencies with a spread to the lower frequencies.

3) Chronic rhinitis rests on physical findings of mucous membrane irritation following exposure to the Alcolac odors and emissions.

4) Liver dysfunction including abnormal LDH, abnormal iron metabolism, abnormal porphyrin metabolism, rests on abnormal results of the tests of these functions of the liver. The heptacarboxylic porphyrin [the cascade at the seventh level of porphryn metabolism] was abnormal, and the coproporphyrin to uroporphyrin ratio was slightly abnormal. The LDH enzyme was abnormally elevated, and suggests liver damage. The serum iron was also abnormal. Since the porphyrins use iron in the production of their chemical and in turn produce an enzyme system, an abnormality in serum iron reflects the abnormal porphyrins and the enzyme system they produce.

5) Early renal insufficiency rests on abnormal laboratory test results. The electrolyte potassium was abnormally low, her uric acid and BUN [blood urea nitrogen] were both elevated. Those conditions represent kidney damage - "the inability of the kidney to get rid of the poisons." They did not represent uremia, but azotemia, a condition short of uremic poisoning.

6) Central nervous system dysfunction including cerebellar damage, diffuse cerebral damage rests on physical findings of increased deep tendon reflexes and the abnormal results of the laboratory tests. A decrease or absence of the deep tendon reflexes are indications of peripheral nerve damage, but increased reflexes indicates damage to the central nervous system. [Persons afflicted by stroke, for instance, show increased, rather than decreased, reflexes.] Thus, the increased deep tendon reflexes represent some evidence of damage to the central nervous system -the brain. There were also findings of cerebellar damage. The cerebellum relates to balance and other important bodily functions. The tests included the tandem walk, the Romberg, and other

procedures to test the balance. The performance of Dorothy Bradley was abnormal. The toxic chemicals emitted by Alcolac, and already identified, affect the central nervous system and the cerebellar function. The neurobehavioral battery administered by Dr. Garron also established abnormality of function and central nervous system dysfunction. The damage is diffuse and affected attention, memory and other components.

The witness, Dr. Carnow, discounted the significance to this subdiagnosis of the prior medical history of a skeletal imaging study on January 4, 1978 at the local hospital of the right parietal bone to explore for possible metastasis. There was concern that there was a tumor, but it was determined, rather, that there was simply a thickening of the parietal bone without pathology. The range of abnormalities found by Dr. Garron - problems in attention, expressive and receptive language, manual dexterity, memory - have nothing to do with the parietal bone, nor do the cerebellar abnormalities found of poor balance and poor taxia. The brain damage was attributed to the effect of the Alcolac emissions.

7) Peripheral nervous system dysfunction rests on findings of weakness in the left leg nerve. The evidence of neuropathy was a decreased vibration in the left ankle and an abnormal response to pain in the left lower leg - the peroneal area. The subdiagnosis was not related to any condition of degenerative joint disease attributable to age because it was the nerve which was found to be abnormal, and not the joint. The degenerative lesion in the left wrist found on physical examination might be a reflection of age, but neither that nor any other musculoskeletal abnormality was diagnosed as related to her exposure to Alcolac.

8) Immune system dysfunction rests on the abnormal test results of the 1984 Bioscience immune panel and of the 1985 Midwest/Wheeler immune panel.

[Bioscience Laboratory Test Results, 1984]

Immune System:
White Blood Cells: slight abnormality of basophils

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune System: | |
| Lymphocytes: | abnormal OKT-4 [helper] cells<br>abnormally elevated OKT-10 cells at 28.3 [normal, 0 to 15]<br>abnormally elevated T-4/T-8 ratio [helper to suppressor cells out of balance] at 4.04 [normal, 1.1 to 2.5] |
| Hematologic System: | abnormal cell size [anisocytosis]<br>abnormal cell shapes [poililocytosis] |

These abnormalities of red blood cell production reflect the abnormal porphyrin metabolism which produces the heme for the red blood cells as well as the abnormal serum iron metabolism, also involved in the production of the red blood cells in the body.

PROGNOSIS: Very guarded.

AMBER CROSS AGE 11 LIFE EXPECTANCY 71.06 YEARS

DIAGNOSIS: Chronic Systemic Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems including:

1) chronic bilateral conjunctivitis
2) chronic rhinitis and sinusitis
3) chronic bronchitis
4) liver dysfunction including:
   a. abnormal porphyrin metabolism
   b. probable chemical hepatitis
5) gastrointestinal dysfunction [possible porphyria crisis]
6) chronic genitourinary disease
7) immune system dysfunction
8) central nervous system dysfunction
9) chronic recurrent dermatitis from probable chemical contact with possible hypersensitivity or allergic components

The witness then explained the data on which the sub-elements of diagnosis rest.

1) Chronic bilateral conjunctivitis rests on red, irritated eyes and blurred vision following exposure to the Alcolac odors and emissions and evident on physical examination.

2) Chronic rhinitis and sinusitis rests on symptoms of dry, burning nose over a long period of time and findings of nose irritation and swelling and inflamed sore throats.

3) Chronic bronchitis rests on symptoms of a productive cough, hemoptysis [coughing up blood] from exposure to the Alcolac chemicals and induced by inhalation of the odors and emissions.

4) Liver dysfunction including abnormal porphyrin metabolism, probable chemical hepatitis rests on abnormal results from the porphyrin, enzyme and prothrombin time tests of the liver functions. It rests also on the physical finding of tenderness over the right upper quadrant, the site of the liver, which suggested a swollen liver. The coproporphyrin, heptacarboxylic porphyrins and porphobilinogens were abnormal. Two liver enzymes were abnormal - the alkaline phosphatase and the LDH. In a growing person, such as eleven year old Amber Cross, the alkaline phosphatase can be somewhat elevated, but her count was twice that of normal. The chemical exposures accounted for that abnormality. A young person, such as Amber Cross, on the threshold of womanhood and menstruation, who has been exposed to the kind of toxic chemicals which affect the P450 enzymes [as do the Alcolac chemicals already identified] is subject to serious crises. The prothrombin time function of the liver also tested abnormally, 44 as to a normal of in excess of 70. It was retested, once again with abnormal result.

5) Gastrointestinal dysfunction [possible porphyria crisis] rests on symptoms of recurrent vomiting, constipation, stomach inflammation and other abdominal and stomach problems

characteristic of porphyria crises which occur when porphyrins "go out of whack." The subdiagnosis was qualified as "possible" because some of these symptoms and manifestations are effects of the nervous system not attributed to the Alcolac emissions. The exposure to the toxic chemicals, however, is the dominant causal factor in her organ dysfunctions.

6) Chronic genitourinary disease rests on the urinalysis result of pus cells in the urine - a manifestation of urinary tract infection. That condition was consistent with the depression of her immune system which renders her more susceptible to viral infection. The prior medical history taken of Amber Cross notes an entry dated February 24, 1978, of dysuria - the difficult or painful discharge of urine. In response to the cross-examination inquiry as to whether that indicated a condition which pre-existed the Alcolac activity, the witness agreed that if Alcolac had emitted no chemical compounds before that date that the genitourinary disease would have to be considered nonattributable to Alcolac. It was the clear evidence, however, that Alcolac undertook test runs as early as 1977 to ensure proper reactor and equipment function when regular production commenced in May of 1978. The medical history of many of the patient plaintiffs [Dr. Carnow observed] disclosed "an abrupt onset of symptoms, some of them early in 1978." The witness concluded that the chronic genitourinary disease was not "a pre-existing lesion" but rather was caused by the toxic chemicals identified and known to target the human kidneys.

7) Immune system dysfunction rests on the abnormal results of the immune panels administered by Bioscience in 1984 and by Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| White blood cells: | abnormally elevated total lymphocytes |
| | abnormally depressed segs [an abnormal ratio since the segs |

| | |
|---|---|
| | are usually higher than the lymphocytes] |
| Hematologic System: | abnormally elevated platelet count at 485,000 |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormal white blood cell count |
| | abnormally elevated OKT-8 [suppressor] cells at 1492 [normal, 140 to 1444] |
| | abnormal - slightly depressed - OKT-4 [helper] cells at 31 [normal 32 to 55] |
| | very abnormal OKT-10 cells at 35.2 [normal, 0 to 15] |
| | abnormal T-4 to T-8 [helper to suppressor] ratio |
| | abnormal elevation in natural killer cells |
| Hematologic system: | abnormally elevated platelet count anisocytosis |
| | abnormal random distribution |

The abnormalities in the immune system had progressed from two in 1984 to six in 1985. Also the abnormality in the platelet count of the red blood system continued and two other abnormalities were tested.

8) Central nervous system dysfunction rests on the abnormal results of the neurobehavioral battery administered to her by Dr. Garron. In that report the examiner rendered the evaluation that the "intellectual and cognitive functioning" of Amber Cross was "unremarkable except for erratic concentration," a "mild impairment in concentration [which] suggests impulsivity, often associated with delinquency and conduct disorders." The examiner commented that these "general characteristics seem to reflect familial instability in Amber's background." In response to the inquiry of the cross-examiner as to whether the subdiagnosis of central nervous dysfunction rests on the neuropsychological report of Dr. Garron, the witness explained that the tester merely conducts the examination and enters a report, but

only he - Dr. Carnow - evaluates the report, as he does all others [laboratory, history, and other data as to each of the patient plaintiffs]. Dr. Carnow amplified that neuropsychological tests report other abnormalities: a moderately poor digit span test, and erratic reaction time test, unusual errors of commission on the coding and underlining test. The entire history of the child, her physical symptoms of vomiting and inflammation of the stomach and defections in other organs following exposure to the Alcolac chemicals, her academic decline from a superior student to a below average student -abnormalities and data Dr. Garron could not have known since he did not have the history information known to Dr. Carnow - require the conclusion that the stress imposed by exposure to the toxic chemicals from Alcolac account for the central nervous system damage found in Amber Cross. That subdiagnosis remains valid, Dr. Carnow explained, albeit the personal disorders exhibited by Joyce Pryor, the mother of Amber Cross may have induced emotional stress in the child.

9) Chronic recurrent dermatitis from probable chemical contact with possible hypersensitivity or allergic components rests on symptoms and episodes of skin irritations and rashes which develop quickly after exposure to the Alcolac odors, vapors and emissions and then spread over her arms, legs, chest and back.

The evaluator concluded that because of her youth, and because of exposure to Alcolac toxic carcinogens, and given the abnormal immune system dysfunction and the abnormal porphyrin metabolism, the probability that Amber Cross would die from some disease of the organ other than cancer is unlikely.

PROGNOSIS: Very guarded to poor.

CLARENCE ELAM AGE 64 LIFE EXPECTANCY 17.89 YEARS

DIAGNOSIS: Chronic systemic Chemical Intoxication caused by Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic chemical conjunctivitis
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic rhinitis and sinusitis
4) abnormal liver metabolism with abnormal porphyrins
5) peripheral neuropathy
6) musculoskeletal dysfunction
7) probable renal insufficiency
8) severe immune dysfunction with suppression
9) possible central nervous system damage

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic chemical conjunctivitis rests on symptoms of reddened, burning and watery eyes following exposure to the Alcolac odors and emissions, with matting of the eyes during sleep at night.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on symptoms of tinnitus and decreased acuity and on the abnormal results of the audiometric test. The right and left ears had a 70 decibel loss at the 3000 frequency cycle and a 50 decibel loss at both the 4000 and 6000 frequency cycles. The neurosensory loss was evaluated as "moderately severe" and very significant.

3) Chronic rhinitis and sinusitis rests on symptoms and findings of mucous membrane irritation and sore throat from the Alcolac odors and emissions.

4) Abnormal liver metabolism with abnormal porphyrins rests on test results of the liver metabolism which disclosed three abnormalities: the coproporphyrins were very elevated at 171 [normal, 10 to 109], the porphobilinogen was abnormal, and the pentacarboxylic [the fifth cascade of porphyrin metabolism] was abnormally high at 7 [normal, 0 to 4]. These represented "a gross disturbance of porphyrins [which] is very worrisome," and amounts to a severe breakdown of a major organ.

5) peripheral neuropathy rests on symptoms of weakness in both arms and the result in the PNVC sensory test which showed an abnormal nerve conduction in the right arm.

6) Musculoskeletal dysfunction rests on symptoms of joint stiffness, leg weakness and joint aches.

7) Probable renal insufficiency rests on symptoms of nocturia and pain in urination and on the abnormally elevated BUN [blood urea nitrogen] and abnormally depressed potassium test results. The BUN, the evaluator explained, is the amount of nitrogenous waste in the body, and when that begins to elevate, it means the body is not getting rid of the waste through the kidneys. It may be a sign of early kidney insufficiency. On cross-examination, the witness was asked to explain the medical history related by Clarence Elam to the examiners under the KU protocol that he has had urgency since childhood due to a small bladder. Dr. Carnow responded that he was given no such history, and in any event, a small bladder might account for urgency, but not nocturia - the abnormal need to empty the bladder. The reason some people have frequency of urination, he explained, is not because the bladder is small or large, but because they do not empty it. Cross-examination noted also a medical record from January of 1971 which indicated a BUN of 21%, with the normal range of 8 to 20%. Dr. Carnow acknowledged that the finding indicated a mildly elevated BUN at that time, but that since the BUN is now 27%, not 21%, the present condition represents a probable renal insufficiency. Dr. Carnow acknowledged that the creatinine was normal when tested, but that means that the kidney is going into failure - a more serious damage than renal insufficiency, the damage the BUN insufficiency connotes. Insufficiency, the witness explained, means the organ does not function as it should, not that "it is in failure or that he is going to die from it."

8) Severe immune dysfunction rests on the abnormal results of the immune panel tests conducted by Bioscience laboratory in 1984 and the Midwest/Wheeler laboratory in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormally depressed at gamma M 61 [normal 80 to 310] |
| Mitogen challenge: | abnormal PHA stimulation net CPM |
| | abnormal PHA stimulation index |
| | abnormal CON A stimulation net CPM |
| | abnormal CON A stimulation index |
| | abnormal PWM stimulation net CPM |
| | abnormal PWM stimulation index |
| White blood cells: | abnormally increased segs |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormality [unspecified] |
| Hematologic system: | abnormal sizes [anisocytosis] |
| | abnormal random distribution width |
| | abnormal mean corpuscular hemoglobin |

These composite tests show that all of the mitogens were abnormal. In addition, the gamma M was quite abnormally depressed. Dr. Carnow concluded that the immune system was "zapped." The results indicate that an abnormal number of the T cells were not programmed to attack an enemy - and so were useless, "just hanging around." He attributed that abnormality to damage to the lymphatic tissue caused by the toxic chemicals from Alcolac which are metabolic poisons. He explained that since the memory T cells live for fifteen or twenty years, when they are poorly programmed and have not been educated to recognize an enemy, they remain useless and contribute to the depression of the immune system. The immune system, Dr. Carnow iterated, was "wiped out."

9) Possible central nervous system damage rests on symptoms of headache, tremor, dizziness and decreased concentration or memory loss.

It was the medical opinion of the expert evaluator that the two severe breakdowns in two major organ systems - porphyrins and the immune system - render Clarence Elam susceptible to pneumonia and many other more serious diseases.

PROGNOSIS: Poor.

BETTY ELAM AGE 59 LIFE EXPECTANCY 25.10

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic bilateral conjunctivitis with probable iritis
2) chronic rhinitis
3) neurosensory hearing loss - mild, with 8th cranial nerve damage
4) chronic chemical bronchitis with obstruction and bronchospasm
5) vascular insufficiency
6) liver dysfunction including:
   a. abnormal lipid metabolism
   b. abnormal protein metabolism - in remission
   c. abnormal prothrombin time - in remission
   d. abnormal iron metabolism
7) genitourinary dysfunction including
   a. possible neurologic deficit
   b. abnormal electrolytes
8) central nervous system dysfunction including
   a. cerebellar dysfunction
   b. severe anxiety (aggravated)
   c. possible cerebral dysfunction
9) peripheral polyneuropathy with progression
10) polymagia and polyarthralgia
11) immune system dysfunction
12) chronic contact dermatitis with probable hypersensitivity component
13) chronic gastrointestinal dysfunction with neurogenic component
14) hematologic dysfunction, possibly secondary to pulmonary insufficiency

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic bilateral conjunctivitis with probable iritis rests on symptoms of red, irritated eyes with blurred vision following exposure to the Alcolac odors and emissions.

2) Chronic rhinitis rests on symptoms of nasal congestion, nosebleeds and irritations of the nose following exposure to Alcolac odors and emissions, and also on the physical finding of an irritated mucous membrane.

3) Neurosensory hearing loss - mild, with 8th cranial nerve damage rests on the abnormal audiometric test results which show a hearing loss at the 4000 and 6000 frequencies. This suggests an early hearing loss with some 8th cranial nerve damage.

4) Chronic chemical bronchitis with obstruction and bronchospasm rests on physical findings of hyperresonance [overinflation] of the lungs - an abnormal difficulty in the expulsion of air from the lungs. The subdiagnosis rests also on symptoms of shortness of breath and hemoptysis [coughing up blood]. The diagnosis of chronic obstructive pulmonary disease from chemical toxicity was entered in the medical records of the Suburban Medical Center in Overland Park, Kansas on September 4, 1981. Dr. Carnow agreed with that diagnosis and attributed that evaluation as well as his own to the inhalation of Alcolac emissions. The prior history shows that Betty Elam has smoked a package of cigarettes a day since she was 18 years of age. Although such a personal regimen could contribute to such a condition of obstruction, Betty Elam does not display the symptoms of such an etiology. Chronic smokers are subject to a class sequence of events, symptoms and stigmata. They begin to cough in the morning, usually in the winter months. Then they cough all year round in the morning, and they bring up phlegm. Then they begin to get serial infections, where they bring up yellow matter. Then they develop obstruction. Betty Elam rarely coughs, and then only when she smells the Alcolac fumes.

Nor does her condition represent emphysema - which is a pathologic diagnosis and means the destruction of lung tissue. The x-ray of the lung was normal. Chronic obstructive lung disease,

on the other hand, means that the person has difficulty expelling air from the lungs - it is obstructed for some reason. The obstruction can result from irritant chemicals - such as epichlorohydrin, the acrylates, the hydrogen sulfide, among the others. The body tries to reject the irritant chemicals, "by closing down." It may cause a spasm of the air passages or swelling of the air passages. In any event, when the body rejects the chemical irritants, it also rejects oxygen - which is very deleterious to the body functions.

5) Vascular insufficiency rests on physical findings of pallor and diminished peripheral pulses in the lower extremities, her feet. These indicate that the blood vessels were narrowed and that the circulation and blood supply into her legs and feet were abnormal. The designation of the Alcolac chemicals as cause for this vascular insufficiency is interrelated to and explained by the phenomenon that these chemicals interfere with the lipid metabolism and may increase the fats in the body very significantly, and so affect the blood vessels of the body. The laboratory tests of the lipid metabolism, in fact, were abnormal.

6) Liver dysfunction including abnormal lipid metabolism, abnormal protein metabolism [in remission], abnormal prothrombin time [in remission] abnormal iron metabolism rests on the abnormal test results for each of these metabolism functions of the liver. There was an abnormal increase in the heptacarboxylic porphyrins and an abnormal ratio between the coproporphyrins and the uroporphyrins. The abnormal ratio may represent a very early abnormality of the level of the prophyrins, thus the disturbance of ratio is significant even in the absence of an abnormal level of one of the porphyrins. The laboratory test disclosed an abnormally depressed iron metabolism, a dysfunction of the enzyme system in which the porphyrins and iron are involved. The prothrombin time was not successfully taken on the first test, and

was not repeated on Betty Elam. The lipid metabolism tested abnormally high for cholesterol at 308 [normal, 140 to 270]. She also had an abnormally elevated LDL [low density lipoprotein, that cholesterol which disfavors health, as contradistinct from HDL, high density lipoprotein, that cholesterol which favors health]. The LDL result was at 243 [normal, 62 to 185], so as to put Betty Elam at "very high risk to coronary artery disease." Her abnormal protein metabolism [presumably inferred from the other results of the liver metabolism functions] was not evident as such and was deemed in remission.

7) Genitourinary dysfunction including possible neurologic deficit, abnormal electrolytes rests on symptoms of difficulty in urination and on the abnormal laboratory test results. There was bacteria in the urine, her creatinine was depressed, as was the potassium.

8) Central nervous system dysfunction including cerebellar dysfunction, severe anxiety [aggravated], possible cerebral dysfunction rests on symptoms of dizziness, decreased concentration or memory loss, depression, nervousness, ataxia [poor balance] and headaches on physical findings of poor balance,and on a test result of 54 on the Zung anxiety scale on a normal of 0 to 44.

9) Peripheral polyneuropathy with progression rests on symptoms of weakness and sensory abnormalities of the limbs and on abnormal test results. The PNVC disclosed an abnormal nerve conduction in the lower extremity [which signifies damage to the sheath of the nerve], and the abnormal EMG signifies axonal degeneration - the actual death of the nerve fiber.

10) Polymalgia and polyarthralgia rests on symptoms of joint stiffness and crepitance, joint aches and muscular pains and cramps.

11) Immune system dysfunction rests on the abnormal results of the immune panels administered by Bioscience laboratory in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulins: | abnormally increased gamma M at 367 [normal, 80 to 310] |
| Mitogen challenge: | abnormally decreased CON A stimulation index |
| | abnormally decreased CON A net CPM |
| White blood cells: | abnormally depressed eosinophils at 0 [normal, 1 to 5] |

[Midwest/Wheeler Laboratory Test Results,1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | no abnormality shown |
| Hematologic system: | abnormally increased mean corpuscular hemoglobin |
| | abnormally increased mean platelet volume |
| | abnormal cell sizes [anisocytosis] |

12) Chronic contact dermatitis with probable hypersensitivity component rests on symptoms of rash, itching, bullae and other skin lesions. The other skin lesions were blisters which means that the chemicals - a number of them alkylating agents, and hence "severe grabbers" - were in such concentration that they literally burned her skin.

13) Chronic gastrointestinal dysfunction with neurogenic component rests on symptoms and history of vomiting, nausea and anorexia.

14) Hematologic dysfunction, possibly secondary to pulmonary insufficiency rests on the symptoms and findings of pulmonary difficulty and lung abnormality and means that some of the changes in her blood supply may be due to the lack of supply of oxygen.

PROGNOSIS: Very guarded to poor.

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic rhinitis and pharyngitis
2) liver dysfunction including:
   a. enlarged, tender liver with probable hepatitis
   b. abnormal lipid metabolism
   c. abnormal iron metabolism
   d. abnormal porphyrin metabolism
   e. abnormal protein metabolism
3) central nervous system damage including
   a. cranial nerve VII damage
   b. cerebellar dysfunction
4) peripheral polyneuropathy
5) immune system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis and pharyngitis rests on symptoms of mucous membrane irritation, swollen nose, soreness and dryness to the throat following exposure to Alcolac vapors and emissions. The chronic pharyngitis rests also on the physical finding of redness to the throat.

2) Liver dysfunction including enlarged, tender liver with probable hepatitis, abnormal lipid metabolism, abnormal iron metabolism, abnormal porphyrin metabolism, abnormal protein metabolism rests on the physical finding of a tender and enlarged liver as well as on the abnormal results of the tests for the several liver metabolism functions. The porphyrin metabolism was abnormal: the coproporphyrins were at 104 [normal, 3 to 56], the porphobilinogen was abnormal, and the coproporphyrin to uroporphyrin ratio was abnormal. The iron metabolism was very low at 47 [normal, 65 to 175]. The cholesterol was abnormally elevated as was the low density lipoprotein [LDL]. The subdiagnosis of abnormal protein metabolism rests on tests of the thyroid which show a depressed T3 uptake and an abnormally depressed free thyroxin [FT]

index. These abnormalities, although of the thyroid, indicated not a hypothyroidism - but that "something was happening in the liver." It signified an abnormality of thyroid binding globulin, a deficiency in the protein metabolism function of the liver.

The examiner, CCA, suggested to Malva Gehlken that the tests be followed up and, in fact, a month later the thyroid panel was tested again by a pathology laboratory in Columbia, Missouri [under whose auspices is not shown]. The results of the successive tests of the thyroid panel were reported as normal. The witness could not explain the disparate results except to observe that exacerbations and remissions are common with chronic diseases. The witness remarked that the Bioscience Laboratory in Chicago, which conducted the first set of tests, "is one of the best labs in the world, and they found these abnormalities." Bioscience, he emphasized, "repeats every test that they do, and I don't know why there is a difference between these two tests." He reaffirmed the subdiagnosis that the tests show that the protein metabolism function of the liver was abnormal.

3) Central nervous system damage including cranial nerve VII damage, cerebellar dysfunction rests on results of the physical examination. The neurobehavioral test results were normal, but the physical findings of the cerebellar function were grossly abnormal: her tandem walk, gait, Rhomberg response, and the other tests of movement and balance were all abnormal. There were also abnormalities of the muscles of the face, indications of damage to the seventh cranial nerve.

4) Peripheral neuropathy rests on the physical finding of decreased deep tendon reflexes, symptoms of sensory loss in the limbs and the laboratory test [PNVC] of abnormal sensory conduction involving multiple nerves.

5) Immune system dysfunction rests on the abnormal results of the immune panel tests administered by Bioscience laboratory in 1984 and Midwest/Wheeler laboratory in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| White blood cells: | abnormally elevated total lymphocytes |
| | abnormally elevated monocytes |
| | abnormally elevated basophils |
| | abnormally depressed segs |
| | abnormally depressed total white cells |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| White blood cells: | abnormally depressed total white cells |
| Lymphocytes: | abnormally elevated OKT-8 [suppressor] cells |
| | abnormal T/4 [helper] to T/8 [suppressor] ratio |
| Hematologic system: | slightly abnormal red cell reduction |
| | abnormally elevated hemoglobin |
| | abnormal cell sizes [anisocytosis] |

These test results of depressed white cell counts, elevated lymphocytes and depressed segs signify a suppression of the immune system. The diseased liver, the severe disturbance of the porphyrin system, and the suppression of the immune system - particularly - rendered her survival questionable.

PROGNOSIS: Poor.

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DAINIE LANDON AGE 74 LIFE EXPECTANCY 11.26 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ system, including:

1) chronic rhinitis and laryngitis with nasal hemorrhage
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic asthmatic bronchitis [aggravated] with chemical component
4) liver dysfunction including:

a. abnormal iron metabolism
b. abnormal porphyrin metabolism

5) genitourinary dysfunction including:
a. abnormal phosphorous metabolism
b. hyperuricemia

6) central nervous system dysfunction
7) peripheral polyneuropathy
8) reproductive system dysfunction
9) immune system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis and laryngitis with nasal hemorhage rests on symptoms and findings of mucous membrane irritation and swelling to the nose, soreness and burning of the throat following exposure to odors and other Alcolac emissions, and recurrent nosebleeds with spurts of blood.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on the abnormal audiometric test results which show bilateral hearing loss at the 3000, 4000 and 6000 cycle frequencies. These findings are characteristic of damage to the 8th cranial nerve.

3) Chronic asthmatic bronchitis [aggravated] with chemical component rests on symptoms of shortness of breath and cough and on abnormal test results of the pulmonary function. The forced vital capacity test result was abnormal at 63% of the total instead of the normal, between 70% and 75%. The medical history shows a diagnosis in 1973 for an emphysematous bleb in the upper right lobe. The witness gave opinion, nevertheless, that the diagnosis could not have been accurate since the present x-ray discloses no such irregularity. An emphysemous bleb means destruction of the lung tissue - a condition which does not "go away." The examination and tests reveal no condition of emphysema at the present time.

4) Liver dysfunction including abnormal iron metabolism, abnormal porphyrin metabolism rest on the abnormal test results of these functions of the liver. There were two porphyrin abnormalities: the coproporphyrins were quite elevated at 124 [normal, 10 to 109] and the porphobilinogen was quite elevated at 2.3 [normal, 0 to 1.5]. The iron metabolism was depressed. That abnormality interrelated with the abnormal porphyrin metabolism as well as with the abnormal hematologic findings.

5) Genitourinary dysfunction including abnormal phosphorous metabolism, hyperuricemia [excess uric acid] rest on symptoms of nocturia and abnormal test results. The urinalysis was abnormal, the uric acid was elevated at 9.1 [normal, 4 to 8.5] and the phosphorous was depressed.

6) Central nervous system dysfunction rests on findings of the neurologic examination which indicate damage to the 7th cranial nerve, the nerve which controls the muscles of the face and expression - in his case, a left central facial weakness. There was also an intention tremor, an abnormal neurologic condition. The past history of a "lazy left eye," Dr. Carnow explained, did not impinge on the subdiagnosis that the central nervous system dysfunctions found were caused by exposure to the Alcolac chemicals. The eyes are controlled by the second, third, fourth and sixth cranial nerves. The seventh controls the expressions of the face, and that is the cranial nerve the neurologic examination found damaged.

7) Peripheral neuropathy rests on symptoms of weakness in the limbs, and findings of sensory abnormalities and decreased deep tendon reflexes on physical examination.

8) Reproductive system dysfunction is not explained. The patient plaintiff Dainie Landon could not produce a specimen for fertility analysis, and so there is no basis in the evidence for subelement 8 the DIAGNOSIS delineates.

9) Immune system dysfunction rests on the abnormal results of the immune panels administered by Bioscience laboratory in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormally elevated gamma A globulins |
| | abnormally depressed gamma M globulins |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally elevated OKT-10 [immature lymphocyte] cells |
| | abnormal T/4 [helper] to T/8 [suppressor] cells |
| | very abnormally elevated [HNK-1 natural killer] cells at 19% [normal, 3.8% to 9.6%] |
| Hematologic system: | decreased cell pigment [hypochromasia] |
| | moderately abnormal sizes [anisocytosis] |
| | abnormally elevated mean corpuscular hemoglobin |

The abnormal increase in the OKT-10 cells signifies immature lymphocytes which have not been properly programmed.

PROGNOSIS: Very guarded to poor.

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MARY LANDON AGE 71 LIFE EXPECTANCY 15.30 YEARS

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DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) recurrent conjunctivitis with possible iritis
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic chemical bronchitis [asthma by history]
4) cardiac dysfunction
5) abdominal cancer
6) liver damage including:
   a. abnormal protein metabolism
   b. chemical hepatitis
   c. abnormal porphyrin metabolism
7) chronic genitourinary dysfunction with recurrent infection
8) central nervous system damage with cerebral dysfunction
9) peripheral neuropathy
10) recurrent skin damage with acute chemical burns

11) severe immune suppression [aggravated by radiation therapy]

The witness then explained the data on which these components of subdiagnosis rest.

1) Recurrent conjunctivitis with possible iritis rests on symptoms of blurred vision and red, irritated eyes following exposure to the Alcolac odors and emissions.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on the abnormal results on the audiometric tests at virtually every cycle, increased at the high frequencies. The hearing loss was to both ears. On the right, there was a 55 decibel loss at the 6000 cycle, a 40 decibel loss at the 4000 cycle, a 40 decibel loss at the 3000 cycle, and a 30 decibel loss at the 1000 cycle. On the left, the loss at each frequency was comparable and extended to the 500 cycle where there was a 35 decibel loss.

3) Chronic chemical bronchitis [asthma by history] rests on symptoms of chest heaviness and cough productive of phlegm and blood. The [asthma by history] presumably alludes to a datum from the narrative given by the patient plaintiff during the pre-examination interview, but that parenthesis is not explained.

4) Cardiac dysfunction rests on symptoms of chest pain, arrhythmia and on the physical finding of an abnormal pulse rate.

5) Abdominal cancer rests on a diagnosis of colon cancer on February 15, 1984, and the surgical removal of that malignancy at the Bothwell Memorial Hospital in Sedalia, followed by a regimen of cobalt therapy. The diagnosis and surgery were accomplished when Mary Landon presented herself at the Sedalia hospital with complaints of stomach pain and blood in the stool and changed bowel habits over the past two years. It was the opinion of Dr. Carnow, to a reasonable medical certainty, that the exposure to the Alcolac toxicants caused the cancer in Mary Landon. That

opinion rests on the carcinogenic qualities of a number of the chemical compounds emitted by Alcolac, the past personal, occupational and environmental history of Mary Landon, and the concentration and duration of the exposures to those toxic substances.

Mary Landon and her husband Dainie lived on a tract immediately west and directly across the road from the entrance to Alcolac. They commenced to notice odors and fumes in the summer of 1978 which gave off the scent of rotten eggs and sulphur. These odors and emissions were from Alcolac and came regularly. They caused her headaches, stomach upsets, coughs with blood in the sputum, chest aches, a fast heart beat, and a congeries of other immediate physical symptoms and discomforts. They began to keep a diary of the Alcolac emissions with descriptions of the symptoms they induced. They maintained the history until they removed to a mobile home on May 8, 1984, to escape the odors and emissions. They record episodes of foam storms from Alcolac which came onto their property and caused her swelling and blisters upon touch. In April of 1981, she consulted a physician in Columbia for recurrent episodes of nausea, nervousness, and dizziness from which she passed out. He discontinued her numerous medications, except for thyroid, but her symptoms continued and worsened. Many of them were directly related to bad odors. She improved on days when there were no odors from Alcolac. Twice after 1978 she was hospitalized when she passed out.

Her pre-Alcolac medical history included a hospitalization for anemia in 1942. Her spleen was removed and the anemia did not recur. Her father had anemia and an enlarged spleen all his life. Thirteen other members of the family had the spleen removed. It was her understanding, Mary Landon testified on cross-examination, that the anemia represented a condition of pre-leukemia, and that was the reason the spleen was removed. The medical records show, rather - as Dr. Carnow explained - that the

anemia was a spherocytosis, a genetic abnormality of the red blood cell whereby the cells become damaged as they pass through the spleen. The remedy is to remove the spleen. It was from that congenital anomaly that the condition of anemia was caused, and it was to allay that anomaly that the spleen was removed from Mary Landon - and the other family members, and not to allay leukemia. The congenital anemia, therefore, no more than that two brothers died of cancer, Dr. Carnow explained, could be deemed a contributing cause to the cancer to Mary Landon by the Alcolac carcinogens.

The cancer found and excised in 1984 was in the gastrointestinal tract. A series of x-rays of the lower tract was done in 1981, and no abnormality or cancer was found. The examination in 1984 did disclose a villous adenoma - a noncancerous lesion. It was not known how long that was present or whether that also developed from the exposure to the Alcolac chemicals, or whether the chemicals converted that benign condition into a cancer. The subdiagnosis of abdominal cancer caused by the Alcolac toxic chemicals did not rest on any such surmise. It rests rather on the carcinogenic properties of epichlorohydrin, glycidyl ether and dimethyl sulfate - toxic compounds used and emitted by Alcolac throughout the period of operations since May of 1978.

6) Liver damage including abnormal protein metabolism, chemical hepatitis, abnormal porphyrin metabolism rests on the abnormal results of laboratory tests of these functions of liver metabolism. The porphyrins were abnormal - a uroporphyria [advanced disease] and coproporphyrin also quite elevated at 98 [normal up to 56]. The enzymes were abnormally elevated - the SGOT, LDH and alkaline phosphatase. These are enzymes seen when the liver is inflamed and damaged. The abnormal increase in the alkaline phosphatase means that the liver damage is more chronic.

The condition represents a chemical type of hepatitis. The abnormality in the protein metabolism was manifested by an elevated gamma globulin.

In response to cross-examination, Dr. Carnow acknowledged that the alkaline phosphatase and SGOT values from the laboratory tests of February 21, 1984 conducted at the time of the abdominal cancer surgery were within normal limits. He acknowledged also that the radiation therapy following the surgery "may well have contributed to the enzyme abnormalities," although not to the other abnormalities of the liver. He concluded, nevertheless, "since the radiation was secondary to a cancer which I feel was caused by exposure to cancer-producing chemicals," the enzyme abnormalities were a consequence causally related to the exposure.

7) Chronic genitourinary dysfunction with recurrent infection rests on prolonged symptoms of pain on urination, frequency of urination, nocturia, and on the laboratory test results of depressed sodium, depressed potassium and abnormal urinalysis. The symptoms alone suggested renal dysfunction. The witness acknowledged, as before, that the post-operative radiation could account for the abnormal test results, since the pre-radiation laboratory tests of these genitourinary elements were normal. He asserted once again that since the cancer was caused by the Alcolac chemicals, and since the radiation was secondary to that cause, the genitourinary dysfunction - as manifested by the laboratory tests - was a direct consequence of the exposure. He iterated, that the test results apart, the recurrent and prolonged symptoms alone suggested renal dysfunction.

8) Central nervous system damage with cerebral dysfunction rests on symptoms of dizziness, decreased concentration, nervousness, sleep disorders, tremor and headache, on the physical finding of increased deep tendon reflexes and on the abnormal

results of the neurobehavioral battery. The neuropsychological tests disclosed a mild deficit in recent memory - a suggestion of mild, diffuse brain dysfunction.

9) Peripheral neuropathy rests on abnormal results in the PNVC, Motor and PNVC, Sensory tests. They demonstrated a slowing of the motor and sensory nerves in the lower left extremity - the limb measured.

10) Recurrent skin damage with acute chemical burns rests on symptoms of itching, burning to the skin, bullae [blisters] and skin infections following exposure to the Alcolac odors and emissions.

11) Severe immune suppression [aggravated by radiation therapy] rests on the abnormal results of the immune panel tests conducted by Bioscience laboratory in 1984.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormal G globulin |
| Mitogen challenge: | grossly abnormal PHA stimulation net CPM |
| | grossly abnormal PHA stimulation index |
| | grossly abnormal CON A stimulation net CPM |
| | grossly abnormal CON A stimulation index |
| | grossly abnormal PWM stimulation net CPM |
| | grossly abnormal PWM stimulation index |
| White blood cells: | abnormal increase in total lymphocytes |

The immune panel test at Bioscience was conducted about six months after Mary Landon concluded the regimen of 40 cobalt treatments post surgery. The results demonstrated grossly abnormal mitogens, as well as the other abnormalities - and manifested as a "very severely depressed" immune system. Dr. Carnow deemed it "impossible to say" how much of that manifestation was from exposure to the Alcolac chemicals and how much from the secondary

radiation. It was his opinion from the full history, symptoms and test results that exposure to the multiple Alcolac carcinogens depressed the immune function, eventuated in the cancer, and that the radiation further depressed the immune system. Thus, the effects of the radiation are in the sequence of causation. He rendered opinion that given the cancer cells - still subsistent - the chemical hepatitis, abnormal porphyrin metabolism, and a very severely depressed immune system, "her longevity will be very limited."

PROGNOSIS: Negative.

Mary Landon died in May of 1986 while the appeal pended.

JOHN LAWRENCE AGE 62 LIFE EXPECTANCY 19.34 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic conjunctivitis
2) severe bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic rhinitis
4) cardiac dysfunction
5) liver dysfunction including:
   a. abnormal lipid metabolism
   b. abnormal protein metabolism
   c. abnormal porphyrin metabolism
   d. abnormal iron metabolism
6) reproduction system dysfunction with abnormal sperm analysis
7) peripheral polyneuropathy
8) polyarthralgia
9) severe immune system depression
10) chronic chemical dermatitis with probable hypersensitivity state

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic conjunctivitis rests on symptoms of red, irritated, blurred and watery eyes following exposure to the Alcolac odors and emissions.

2) Severe bilateral neurosensory hearing loss with 8th

cranial nerve damage rests on the abnormal results of the audiometric tests. He was almost totally deaf at a number of frequencies. On the right ear was a 85 decibel loss at 6000 cycles, a 95 decibel loss at 4000 cycles, a 95 decibel loss at 3000 cycles, a 40 decibel loss at 2000 cycles, a 30 decibel loss at 1000 cycles and a 30 decibel loss at 500 cycles. On the left ear, the decibel loss was comparable at each frequency cycle. The findings signify destruction of the 8th cranial nerves, a condition not meliorated by a hearing aid. The hearing loss pattern was characteristic of chemical intoxication of the 8th cranial nerves, and uncharacteristic of loss of hearing due to age [presbycusis].

3) Chronic rhinitis rests on symptoms of irritated nose and sneezing following exposure to the Alcolac odors and emissions and on the physical finding of mucous membrane irritation.

4) Cardiac dysfunction rests on the physical finding of diminished peripheral pulse and an abnormal conduction on an EKG tracing.

5) Liver dysfunction including abnormal lipid metabolism, abnormal protein metabolism, abnormal porphyrin metabolism, abnormal iron metabolism rest on the abnormal results of the tests of these functions of liver metabolism. The lipid metabolism showed an abnormally elevated triglycerides and an abnormal phenotype [hyperprebeta lipoproteinemia, an abnormal pattern of body fats]. The iron metabolism was depressed - which represents an abnormality in the enzyme system.

There was no explanation, by test result or other diagnostic basis, for the abnormal protein metabolism or abnormal porphyrin metabolism components of the subdiagnosis - and we assume that entry of those manifestations within the Chronic Systemic Chemical Intoxication diagnosis rendered for John Lawrence was mistaken.

6) Reproduction system dysfunction with abnormal sperm analysis rests on the results of the fertility test. John Lawrence and Gwendolyn Lawrence, along with Betty Elam and Charlotte Phillips were the four among the patient plaintiffs who were examined and tested twice at CCA, once in 1983 and then in 1984. On the 1983 occasion, John Lawrence was not able to produce a specimen, so there was no fertility analysis. On the 1984 occasion, the specimen produced showed a markedly depressed count - a total count of 14.8 million [normal, above 60 million]. Thirty-five percent of the sperm were abnormal, a percentage higher than is normally found. The very low overall count as well as the very high count in abnormal morphology unquestionably constitutes a reproductive system dysfunction, the evaluator concluded. It represents as well, Dr. Carnow explained, damage to the testicular function. That derangement of function was explained as the action of epichlorohydrin and some of the other Alcolac toxic chemicals on the P450 enzyme system. That enzyme system is particularly active in the functions of high energy systems such as the brain, kidney and formation of the sperm. The abnormality of function caused to the P450 enzyme system by these chemicals affects the reproductive function. In addition, chemicals such as epichlorohydrin are alkylating agents which grab tissue - protein - and destroy the cells. Dr. Carnow testified that testicular atrophy from these chemicals has been demonstrated in animal studies.

The witness responded to cross-examination that the medical record of July 12, 1977 of a benign prostatic hypertrophy merely indicates an enlarged prostate and has no significance for the reproductive system. The only function of the prostate in reproduction is to secrete an alkaline fluid to nurture the sperm for the three days it takes for the journey to fertilize the egg.

The production of the sperm takes place in the testicle, not in the prostate, thus an enlarged prostate does not bear on the subdiagnosis of reproductive system dysfunction.

7) Peripheral polyneuropathy rests on the symptoms of leg cramps and other sensory abnormality as well as on the finding of abnormally decreased deep tendon reflexes upon the second physical examination in 1984. The first examination of the deep tendon reflexes in 1983 was normal, as were the nerve conduction velocity tests [EMG]. The second EMG was also normal, but on the second physical examination, the deep tendon reflexes at both biceps, both triceps and both wrists were abnormal. That suggested that John Lawrence had developed these peripheral neuropathic findings. That the two tests were conducted by two different examiners, the first by an occupational medicine physician and the second by a neurologist, does not bear on the validity of each finding, although different, where the examiners - as here - are both skilled.

8) Polyarthralgia rests on symptoms of recurrent muscle pains after exposure to the Alcolac odors and emissions.

9) Severe immune system depression rests on the abnormal results of the immune panel tests conducted by Bioscience laboratory in 1984 and Midwest/Wheeler laboratory in 1985.

[Bioscience Laboratory Test Results 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormal gamma M at 76 [normal, 80 to 310] |
| Mitogen challenge: | abnormal PHA stimulation index at 102 [normal, over 130] |
| | abnormal PHA stimulation net CPM at 25,000 [normal, over 62,000] |
| | abnormal CON A stimulation index at 37 [normal, over 40] |
| | abnormal CON A stimulation net CPM at 9000 [normal, over 12,000] |
| | abnormal PWM stimulation index at 18 [normal, over 20] |
| | abnormal PWM stimulation net CPM at 4400 [normal, over 8000] |

White blood cells: abnormal basophils, slightly increased

[Midwest/Wheeler Laboratory Test Results, 1985]

Immune system:

Lymphocytes: abnormal increase of NKH-1 [natural killer] cells

Hematologic system: abnormal increase in platelet volume

The first examination at CCA revealed an immune system only "slightly abnormal." The second examination a year later found the immune system "completely zapped." The mitogen challenges were all abnormal, the immunoglobulin was abnormal, and additionally, the subsequent Midwest/Wheeler results in 1985 [which tested a different function] disclosed the additional result of an abnormal increase in the natural killer cells of the immune system. That system Dr. Carnow described as effectively "wiped out." The destruction of the immune systems of both John Lawrence and Gwendolyn Lawrence [as we soon note] is explained by the heavy exposure over the period of years to the Alcolac toxic chemicals by persons in the near vicinity.

10) Chronic chemical dermatitis with probable hypersensitivity state rests on symptoms of rash, itch, hives [bullae], skin infections and other skin lesions following exposure to the Alcolac odors and emissions.

The evaluator concluded that the damage to the liver, extensive peripheral neuropathy and severe immune system depression suggest continued deterioration and result in a considerably shortened life.

PROGNOSIS: Poor.

GWENDOLYN LAWRENCE AGE 64 LIFE EXPECTANCY 20.92 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) mucous membrane irritation of the eyes, nose and throat
2) chronic chemical bronchitis
3) cardiac dysfunction with arrythmias
4) chronic gastroenteritis (central nervous system component)
5) Liver dysfunction including
   a. possible chemical hepatitis with abnormally elevated LDH
   b. abnormal iron metabolism
   c. probable abnormal porphyrin metabolism
6) possible obstructive uropathy with episodic bleeding
7) central nervous system dysfunction including:
   a. cerebellar damage
   b. cerebral damage with deterioration
   c. severe anxiety
8) peripheral polyneuropathy
9) hypothyroidism
10) musculoskeletal dysfunction with myositis
11) severe suppression with marked deterioration

The witness then explained the data on which these components of subdiagnosis rest.

1) Mucous membrane irritation of the eyes, nose and throat rests on symptoms of red, irritated eyes with blurred vision, irritated nose and nosebleeds with soreness of the nose, burning and irritation of the throat following exposure to the Alcolac odors and emissions.

2) Chronic chemical bronchitis rests on symptoms of shortness of breath, constant hacking cough which produces sputum streaked with blood. As is characteristic of a chronic disease, the intensity of the symptoms as well as of the discharge varied from time to time.

3) Cardiac dysfunction with arrythmia rests on symptoms of chest pain and arrythmia following exposure to the Alcolac odors. It rests also on the physical finding of abnormal heart rhythm, a condition of heart muscle irritation induced by the Alcolac toxic agents.

4) Chronic gastroenteritis [central nervous system component] rests on symptoms of vomiting and nausea which started in late 1978, became most intense in 1982, and then less frequent during that year. The symptoms followed after the inhalation of the Alcolac odors for several days. That manifestation of gastroenteritis was related - Dr. Carnow concluded - to an autonomic [central] nervous system component which caused her to vomit. In response to the cross-examination that a medical record of 1974 suggests a subsistent hiatal hernia, the witness responded that a vomiting upon the inhalation of a toxic chemical is "a very common reaction when the central nervous system is assaulted by a toxic agent." It is not a symptom of hiatal hernia, which simulates an ulcer after a person who has one ingests food. It relates neither to smell nor to severe vomiting - the symptoms Gwendolyn Lawrence experienced and described.

5) Liver dysfunction including possible chemical hepatitis with abnormally elevated LDH,.abnormal iron metabolism, probable abnormal porphyrin metabolism rests on the abnormal laboratory test results of these functions of the liver. There was one abnormal porphyrin: the heptacarboxylic was depressed at 1 [normal, 3 to 9]. The enzyme LDH was abnormally elevated. The iron metabolism was quite depressed at 38 [normal, 65 to 175].

6) Possible obstructive uropathy with episodic bleeding rests on symptoms of frequent urination in nocturia, foul urine smell and on the abnormal urinalysis.

7) Central nervous system dysfunction including cerebellar damage, cerebral damage with deterioration, severe anxiety rests on symptoms of dizziness, sleep disorders, poor balance [ataxia], and headache and on findings on physical examination and on the abnormal results of the Zung anxiety scale. Gwendolyn Lawrence was among those examined at CCA in 1983. The neurological tests on that occasion disclosed abnormal tandem walk and

position sense - cerebellar dysfunctions. The subsequent test in 1984 was normal for those functions, but her symptoms continued. She still staggered when she walked and could no longer ride a bicycle because of imbalance. Her Zung anxiety scale was moderately high at 53 [normal, 0 to 43].

8) Peripheral polyneuropathy rests on symptoms of sensory abnormalities of tingling, numbness, pins and needles in the hands and of weakness, on the finding on physical examination in 1984 at CCA of weakness in the hand, and abnormal nerve conduction tests, both motor and sensory on the lower left [PNVC, motor, PNVC, sensory]. These demonstrated depressed nerve function at the lower extremity. The examinations, symptoms and tests conducted in 1984 demonstrated "a very considerable deterioration of her peripheral nervous system" since the 1983 evaluation.

9) Hypothyroidism rests on abnormal T3 uptake and FT index tests of the thyroid component of the endocrine system. This manifestation represented both a thyroid binding globulin abnormality, but also some hyperthyroidism - as the physical finding of a slightly enlarged thyroid suggests.

10) Musculoskeletal dysfunction with myosis rests on symptoms of muscular cramps, joint stiffness and aches following exposure to the Alcolac odors as well as on an abnormal CPK test result. CPK is an enzyme which becomes abnormal when acute inflammation of the muscle is present. Her CPK was abnormally elevated at 99 [normal, up to 60].

11) Severe immune suppression with marked deterioration rests on the abnormal results from three series of tests, Bioscience laboratory in 1983, Bioscience laboratory in 1984, Midwest/Wheeler in 1985:

[Bioscience Laboratory Test Results, 1983]

Mitogen challenge: abnormally low PHA stimulation net CPM at 30,785 [normal, over 62,000]

abnormally low PHA stimulation index at 71 [normal, over 130]
abnormally low CON A stimulation net CPM at 8532 [normal, over 12,000]
abnormally low CON A stimulation index at 20 [normal, over 40]
abnormally low PWM stimulation net CPM at 7504 [normal over 8000]
abnormally low PWM stimulation index at 18 [normal, over 20]

[Bioscience Laboratory Test Results, 1984]

Immune system:

Mitogen challenge:
abnormally low PHA stimulation net CPM at 1200
abnormally low PHA stimulation index at 6
abormally low CON A stimulation net CPM at 144
abnormally low CON A stimulation index at 2
abnormally low PWM stimulation net CPM at 359
abnormally low PWM stimulation index at 2

White blood cells: abnormally low total lymphocytes

[Midwest/Wheeler Laboratory Test Results, 1985]

Hematologic system:
abnormally depressed mean corpuscular hemoglobin
abnormally elevated platelet volume
anisocytosis [abnormal shapes]
hypochromasia [abnormal color]

Dr. Carnow interpreted these laboratory results as an immune system in progressive and "terrible" deterioration, a system in "total suppression" - even worse than that of Mary Landon after the course of radiation for her cancer. The antigens introduced to the immune system cells on the mitogen challenges, Dr. Carnow explained, are expected to be seen as enemies and attacked. The PHA antigen is one that the killer cells attack - so the PHA count suggests a virtually nonexistent killer cell population [at 1200 with the normal over 62,000]. The poke weed

mitogen [PWM] tests the B lymphocytes, and the count of the response to that antigen suggests a lack of function of those cells. CON A generally relates to the helper cells function, and the stimulation net at 144 [normal over 12,000] and stimulation index at 2 [normal, over 40] demonstrates that the entire immune system is irreparably suppressed.

PROGNOSIS: Poor.

GLENN MILLER AGE 54 LIFE EXPECTANCY 25.51 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic bilateral conjunctivitis
2) chronic rhinitis
3) liver dysfunction including:
   a. abnormal lipid metabolism
   b. abnormal porphyrin metabolism including coproporphyrin and porphobilinogen
4) genitourniary system dysfunction with infection
5) reproductive system dysfunction
6) central nervous system damage with diffuse brain dysfunction
7) peripheral neuropathy
8) immune system dysfunction with suppression

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic bilateral conjunctivitis rests on recurrent symptoms of red, irritated eyes following exposure to the odors and fumes from Alcolac, and on reddened conjunctiva on physical examination. The physical findings are indications that the chronic assault of these toxic substances has undermined the power of the body to repair the damage.

2) Chronic rhinitis rests on symptoms of nosebleed and on the findings of reddened, swollen mucosa and of exudate in the nose upon physical examination.

3) Liver dysfunction including abnormal lipid metabolism, abnormal porphyrin metabolism including coproporphyrin and porphobilinogen rests on abnormal results of laboratory tests of those liver metabolism functions. There were two abnormal porphyrins: the coproporphyrin was quite high at 130 [normal, up to 110], and the porpobilinogen was increased at 2.3. The triglycerides test was high and represents an increase in abnormal lipid metabolism.

4) Genitourinary system dysfunction with infection rests on a urinalysis which disclosed pus cells. The test was repeated by a private physician, which also disclosed infection, and Glenn Miller was bedridden and treated.

5) Reproductive system dysfunction rests on the fertility analysis results of multiple abnormalities in sperm count, percent activity and morpholgy. The activity of the sperm after 30 minutes was slightly decreased from 80% to 75%. The count at 39.6 million was low even as compared with the marginal minimum count of 60 million. Also, 45% of the sperm were abnormal - an unduly large count.

6) Central nervous system damage with diffuse brain dysfunction rests on abnormal results of the neurobehavioral battery. It is characteristic of toxic depression of the brain function [although toxic agents can induce a focal lesion] that the damage is not localized. The pattern disclosed by the neurobehavioral battery is of perceptional deficits, attentional deficits, perceptual motor function and problem solving - clinically significant indicia of diffuse brain malfunction.

7) Peripheral neuropathy rests on symptoms of weakness of the limbs and sensory abnormalities - tingling and numbness - and on the findings on physical examination of absent deep tendon reflexes and sensory abnormalities.

8) Immune system dysfunction with suppression rests on the abnormal results of the tests conducted by Bioscience laboratory in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | | |
|---|---|---|
| Immune system: | | |
| | Mitogen challenge: | an abnormal PHA stimulation net CPM at 59,000 [normal, over 62,000] |
| | White blood cells: | abnormally depressed eosingophil at 0 |
| | | abnormally depressed white cell count at 4300 [normal, 4500 to 11,000] |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | | |
|---|---|---|
| Immune system: | | |
| | Lymphocytes: | abnormally elevated OKT-10 cells at 27 [normal, 0 to 15] |
| | | abnormally elevated NKH-1 cells at 14.9 [normal, up to 9.6] |
| | Hematologic system: | abnormally elevated mean corpuscular hemoglobin |
| | | abnormally elevated mean platelet volume |

The evaluator explained that NKH-1 cells, killer cells, also measure other suppressor cells, as does the OKT-10, both show marked elevation, and hence present a "very abnormal" immune dysfunction. The patient plaintiff, Glenn Miller, was advised by CCA to avoid further exposure to chemicals, take care about exposure to viral or fungal infections since the low T-lymphocyte function the tests disclosed make him more susceptible to such disease. He was also warned to avoid certain drugs which might precipitate a porphyria attack.

It was elicited on cross-examination that in November of 1980 the patient plaintiff Glenn Miller was received at the Bothwell Memorial Hospital in Sedalia with complaints of headache and nausea from the use of bug spray at the school where he worked. A later entry in the medical record book indicated that his sense of well-being was restored when the exposure ceased. Dr. Carnow was asked whether the occupational exposure to the bug

spray explained or impinged on any of the problems diagnosed for Glenn Miller. The response was that the symptoms described in the hospital records were those from an episode of acute exposure and subsided when the exposure ceased. The bug sprays in the schools serviced by Glenn Miller use pyrethrums or other such substance which, although they may cause headache, are not very toxic. However, the organ damage and disease diagnosed for Glenn Miller - Dr. Carnow explained - are not from a single exposure nor from a chemical agent of only mild toxicity. They are from chronic exposures over many nights and for very long periods of time and by agents of severe toxicity - such as epichlorohydrin. The exposure to the school spray - even for a year and a half, as the medical record indicates - could not account for the multiple organ damage found in Glenn Miller. That is so even if the actual spray chemical and the extent of the exposure were known. His medical history and complaints indicate that Glenn Miller has felt no better even after the exposure to the bug spray ceased. He continues to lack energy and vitality that he enjoyed before the Alcolac emissions, and he continues to relate these and other symptoms to the exposure to the Alcolac toxic agents.

PROGNOSIS: Very guarded.

BERNICE MILLER AGE 54 LIFE EXPECTANCY 29.51 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic bilateral conjunctivitis
2) bilateral hearing loss with probable neurosensory component
3) chronic rhinitis and pharyngitis
4) liver dysfunction including:
   a. abnormal porphyrin metabolism including uroporphyrin, coproporphyrin and porphobilinogen
5) peripheral neuropathy
6) immune system dysfunction
7) hypertension - mild

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic bilateral conjunctivitis rests on symptoms of red, irritated and blurred eyes following exposure to the Alcolac odors and the physical finding of edematous conjunctiva.

2) Bilateral hearing loss with probable neurosensory component rests upon the audiometric test results which were abnormal at every frequency for both ears - except at the 2000 frequency, where the hearing at the right ear was abnormal, but normal at the left ear. The neurosensory loss ranges at 45 decibels at the 3,000, 4,000 and 6,000 cycle frequencies, and a 35 decibel loss at the 500, 1,000 and 2,000 cycle frequencies. The results represent neurosensory loss extended to the lower frequencies - an 8th cranial nerve damage.

3) Chronic rhinitis and pharyngitis rests on symptoms of nose irritations and soreness in the throat following exposure to the Alcolac odors. The chronic rhinitis rests also on the findings upon physical examination of irritated, reddened and swollen mucous membrane with exudate material.

4) Liver dysfunction including abnormal porphyrin metabolism including uroporphyrin, corpoporphyrin and porphobilinogen rests on the multiple abnormal test results of that function of the liver. They show a uroporphyrin at 33 [normal, 4 to 22], a coproporphyrin at 135 [normal, 3 to 56], and a porphobilinogen at 3 [normal, 0 to 1.5] - three abnormalities along the porphyrin cascade. The uroporphyria signifies a disease somewhat advanced. The iron metabolism, a phenomenon related to the porphyrin abnormalities, was quite depressed at 52 [normal, more than 65].

5) Polyneuropathy rests on symptoms of numbness in both hands and weakness, and on the finding on physical examination of an absent deep tendon reflex in the left ankle.

6) Immune system dysfunction rests on the abnormal results of the tests conducted by Bioscience laboratory in 1984 and Midwest/Wheeler laboratory in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormal globulin A |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally depressed OKT-11 [total T] cells<br>abnormally increased OKT-10 [unprogrammed lymphocyte] cells<br>abnormally increased [natural killer] cells |
| Hematologic system: | abnormally increased mean platelet volume |

It was the opinion of the evaluator that the test results of abnormal depression of total T cells and abnormally increased natural killer cells signify a suppression of the helper cells, and hence an imbalance between the helpers and suppressors.

7) Hypertension - mild is a subdiagnosis neither evaluated by the witness nor otherwise explained in the evidence, and - we assume - was mistakenly entered as a diagnosis.

PROGNOSIS: Very guarded to poor.

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JOHN PHILLIPS AGE 58 LIFE EXPECTANCY 22.35 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by Alcolac toxic chemicals with damage to multiple organ systems, including:

1) bilateral cataracts with recurrent acute conjunctivitis
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic chemical bronchitis with obstruction
4) liver dysfunction including:
   a. abnormal porphyrin metabolism
5) central nervous system dysfunction with:
   a. damaged cranial nerves
   b. cerebellar damage
   c. depression
   d. anxiety

e. cerebral damage with particular involvement of the right side of the brain

6) severe far-advanced polyneuropathy

The witness then explained the data on which these components of subdiagnosis rest.

1) Bilateral cataracts with recurrent acute conjunctivitis rests on symptoms of blurred vision, red, irritated eyes following exposure to the Alcolac odors and emissions. The cataracts were diagnosed from the neurobehavioral tests which detected that John Phillips tended to neglect the left side of his visual space and displayed a mild deficit in visual perception. A workup the year before disclosed retrolental opacities not amenable to treatment.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on the abnormal results of the audiometric tests of both ears at the 3000, 4000 and 6000 cycle frequencies. On the right, there is a 65 decibel loss at 3000 cycles, 75 decibel loss at 4000 cycles and 70 decibel loss at 6000 cycles. On the left, there is a 60 decibel loss at 3000 cycles, 80 decibel loss at 4000 cycles and 70 decibel loss at 6000 cycles. The bilateral loss of hearing is quite severe.

3) Chronic chemical bronchitis with obstruction rests on symptoms of shortness of breath upon inhalation of the Alcolac odors and emissions, on findings of wheezing upon physical examination, and on the abnormal result of the pulmonary $FEV_1/FVC$ test. The result of that test was a vital capacity of 51% of the total - which is a marked diminution from the normal and signifies a pulmonary obstruction.

In the attribution of cause of the chronic bronchitis with obstruction to the Alcolac chemicals, the evaluator took into account that John Phillips had smoked a pack of cigarettes a day since he was about 16 years of age, but gave up the practice when

a physician diagnosed his condition as "bronchial obstructive disease with reversible bronchial irritation." He has since smoked a pipe infrequently during the day. The earlier diagnosis describes a difficulty in exhalation because of the spasm caused to the lungs by the irritant material [cigarette smoke]. In fact, as the evaluator acknowledged, John Phillips "does give a classic history of an individual who has chronic bronchitis from cigarettes." It does not, however, show a history of a bronchitis developed into obstructive disease. There was no sputum, no wet coughs, no recurrent infections which mark that progression. He was progressing well until two years of exposure to the Alcolac chemicals, and suddenly is beset with chest problems. The evaluator concluded that although the cigarette use may have contributed to the obstruction, it was not a major factor in the etiology of that disease.

The medical history also reported a complaint to the Veterans Administration Hospital in Columbia on April 24, 1977 of "some shortness of breath on admission." The occasion for the hospital visit was a complaint of dizziness, weakness and "spinning around." Dr. Carnow surmised that it involved "some kind of an acute problem . . . some kind of a Meniere's syndrome." Examination, however, found the lungs completely normal - and hence, whatever that episode meant, it did not involve the lung function and hence does not impinge on the subdiagnosis that the pulmonary obstruction was caused by the Alcolac chemicals.

4) Liver dysfunction including abnormal porphyrin metabolism rests on the abnormal results of the tests of that function of the liver. The coproporphyrins were abnormally elevated at 127 [normal, 10 to 109] and the porphobilinogen is elevated at

1.7 [normal 0 to 1.5]. The liver dysfunction [although not marked on the DIAGNOSIS chart prepared by Dr. Carnow prior to testimony] also included a manifestation of abnormal protein metabolism. The globulins were abnormally depressed.

5) Central nervous system dysfunction with damaged cranial nerves, cerebellar damage, depression, anxiety, cerebral damage with particular involvement of the right side of the brain rests on multiple symptoms, physical findings and abnormal test results. The symptoms which aided subdiagnosis were dizziness, loss of appetite, inability to sleep and headaches. The findings on physical examination were abnormality of the 12th cranial nerve and of the 8th cranial nerve as well as of the cerebellar functions of balance and tandem walk. The Zung anxiety scale was very high at 64 and the Beck depression inventory was also quite severe at 31 [normal, 14-16] - a state of agitated depression. The neurobehavioral battery - a series of 20 procedures which test visual/spatial perceptions and other brain functions - yielded virtually all abnormal results. The neurologist examiner concluded [and Dr. Carnow agreed] that the test results suggest "right hemisphere brain dysfunction." Dr. Carnow commented that the condition of organ depression exhibited by John Phillips [anxiety due to damage to the brain] renders function as to ordinary matters all the more difficult. That tends to explain the persistent marital discord from the insistence by Mrs. Phillips that they move away from the Alcolac disruptions and his refusal to leave the family homestead.

Dr. Carnow discounted any prior medical history to explain the etiology of the central nervous system dysfunction and attributed that manifestation of subdiagnosis entirely to exposure to the Alcolac emissions. The cyst suggested by a VA Hospital CAT scan in 1977 - for instance - was on the left side of the

brain, and so is unrelated to the damage diagnosed to the right side of the brain from the toxic emissions.

6) Severe far-advanced polyneuropathy rests on symptoms of weakness, tingling and cramps in the limbs, on physical findings of sensory abnormalities and on the abnormal results of the EMG, PNVC, motor and PNVC, sensory tests. The upper right extremity and the contralateral lower left extremity were tested. Every nerve tested in those limbs was abnormal. The results showed not only damage to all the nerves, but axonal degeneration - complete destruction of the nerves.

The immune system also tested abnormally, both on the immune panel administered by Bioscience in 1984 and by Midwest/-Wheeler in 1985. Thus, the Bioscience results show a depressed immunoglobulin M at 71 [normal, 80 to 310], elevated white blood cell monocytes and elevated basophils. The Midwest/Wheeler 1985 immune panel shows both an abnormal increase in B cells and an abnormal percent of B cells. These abnormalities were not rendered into a subdiagnosis on the DIAGNOSIS chart. It was the opinion of Dr. Zahalsky, immunology expert that all of the thirty-one plaintiffs [John Phillips included] suffer from a "progressive chemical intoxication of the immune system." That condition of intoxication, he gave as opinion, will subject them to "a variety of diseases, including, but not exclusively neoplastic disease." The formal DIAGNOSIS chart, for whatever reason, does not enter such a diagnosis for John Phillips.

PROGNOSIS: Poor.

CHARLOTTE PHILLIPS AGE 52 LIFE EXPECTANCY 31.34 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic conjunctivitis
2) borderline hearing loss
3) chronic pharyngitis and bronchitis

4) chronic gastritis - severe
5) liver dysfunction including:
   a. abnormal LDH
   b. lipid abnormality - aggravated
   c. abnormal porphyrins
   d. abnormal protein metabolism - possible hypothyroidism
6) chronic esophagitis
7) hypertensive cardiovascular disease with chronic coronary insufficiency
8) genital urinary dysfunction with chronic urinary tract difficulty
9) central nervous system dysfunction with severe anxiety and depression
10) peripheral neuropathy
11) immune system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic conjunctivitis rests on symptoms of redness, irritation and itchiness of the eyes following exposure to the Alcolac odors and emissions.

2) Borderline hearing loss rests on the abnormal results of the audiometric test administered to Charlotte Phillips by CCA in 1984. [She was one of the four who were examined successively by CCA in 1983 and 1984.] The results were slightly abnormal - a 30 decibel loss - at the 3000, 4000 and 6000 frequency cycles. They nevertheless indicate neurologic damage. In 1983, no audiometry was conducted, but a history of noisy employment was noted. There was some indication of hearing loss at the lower frequencies on that occasion. The eardrums were scarred, which indicated a conduction rather than a nerve defect. The cause of the hearing loss noted in 1984, a borderline effect, was attributed to the chemicals because she had made no previous complaint of hearing loss, had not been tested previously by audiometry and the toxic chemicals emitted by Alcolac do damage the 8th cranial nerve. Neurosensory hearing loss may either be noise or chemical induced, but the probability of nerve damage to one exposed to job noise is markedly increased by exposure to chemicals.

3) Chronic pharyngitis and bronchitis rests on symptoms of burning, sore throat, shortness of breath and cough and on the physical finding of cough. The symptoms followed exposure to the Alcolac odors and emissions.

4) Chronic gastritis - severe rests on prolonged symptoms of stomach cramps with the distress of burning sensations and nausea from the odors and emissions.

She was advised by a physician that these symptoms of gastrointestinal distress were attributed to a hiatal hernia diagnosed in 1979. Also, her gallbladder was removed in 1974, but the problems related to that malady did not recur thereafter. The medical records contained the history of these conditions.

The medical history of Charlotte Phillips discloses that in 1974 her gallbladder was removed and in 1979, her symptoms of gastrointestinal distress - also nausea and burning sensations in the stomach area - signified a hiatal hernia. The symptoms relating to the gallbladder did not recur after the surgery, and although a hiatal hernia may explain the subsequent gastrointestinal symptoms Charlotte Phillips described, after the removal of the gallbladder in 1974 there were no significant symptoms until after exposure to the Alcolac chemicals in 1978. The significant array of problems Charlotte Phillips described - epigastric pain, gastroenteritis, nausea, diarrhea, colitis - all began in 1978 after Alcolac commenced to generate the toxic chemicals to which she was exposed. It was also after 1978 that she relates similar reactions from her domestic animals - such as vomiting. It was the opinion of Dr. Carnow, based upon this medical and environmental history, that the distresses already described - epigastric pain and the others - that she did not have before the Alcolac operation in 1978 were attributable to the

exposure to those odors and emissions. Dr. Carnow acknowledged that the hiatal hernia itself was not an effect of the Alcolac chemicals.

5) Liver dysfunction including abnormal LDH, lipid abnormality - aggravated, abnormal porphyrins, abnormal protein metabolism - possible hypothyroidism rests on the laboratory test of these liver functions. The liver enzyme LDH was somewhat elevated at 133 [normal, 62 to 131] and signified some inflammation of the liver. The lipid metabolism was abnormal: the lipoprotein phenotype was abnormal; the tryglycerides were greatly elevated at 301 [normal, 30 to 135]. Two porphyrins were abnormal: the uroporphyrins were elevated at 35 [normal, 4 to 22] and the coproporphyrin to uroporphyrin ratio was abnormal. These abnormalities signify a condition of uroporphyrinuria. The abnormal protein metabolism was demonstrated by the results of the thyroid tests of the endocrine system. The T3 uptake was depressed as was the FT index. Dr. Carnow concluded that those results may represent a low thyroid [hypothyroidism], but that the underlying pathology nevertheless was the abnormal protein metabolism.

6) Chronic esophagitis rests on the symptoms of inflammation of the esophagus from the recurrent vomiting. This condition was attributed to the effect of the Alcolac odors and emissions and not to the previous condition of hiatal hernia for the same reasons ascribed for the causation of the chronic gastritis subdiagnosis component.

7) Hypertensive cardiovascular disease with chronic coronary insufficiency rests on symptoms of pain in the midchest which radiate to the sides of the chest, and on physical findings of abnormal pulse rate and diminished peripheral pulse.

8) Genitourinary system dysfunction with chronic urinary tract difficulty rests on an abnormal urinalysis, with elevated pus cells and bacteria and recurrent urinary infection.

9) Central nervous system dysfunction with severe anxiety and depression rests on symptoms of dizziness, depression and headache and on the abnormal results of the Zung anxiety scale, neurobehavioral battery and Beck depression inventory. On the neurological tests, she displayed a mildly unreliable concentration, a mildly slow digit vigilance response and a mildly slow bilateral finger-tapping. The anxiety was moderate at 26, on a Beck scale of 0 to 10. The depression was high at 63, on a Zung scale of 0 to 44. These abnormalities, the evaluator interpreted to be manifestations of organic origin interrelated with the chemical component. The hyperexcitability at the high level of anxiety combined with the state of depression found in Charlotte Phillips was particularly ominous to a restoration of a healthy state.

10) Peripheral neuropathy rests on symptoms of sensory abnormalities as well as cramps and tingling in the limbs, and on the physical finding of the decreased or absent deep tendon reflexes.

11) Immune system dysfunction rests on the abnormal results of the immune panels administered by Bioscience laboratory in 1984 and Midwest/Wheeler laboratory in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormally increased globulin G at 1510 [normal, 700 to 1500] |
| White blood cells: | abnormally elevated monocytes |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally depressed white blood cell count |
| Hematologic system: | abnormally increased mean platelet volume |

PROGNOSIS: Very guarded to poor.

DANIEL PRYOR AGE 35 LIFE EXPECTANCY 42.63 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic rhinitis and conjunctivitis
2) chronic chemical bronchitis with restrictive and obstructive defects; and recurrent hemorrhage
3) chronic gastritis
4) liver dysfunction with:
   a. abnormal lipid metabolism
   b. abnormal protein dysfunction
5) reproductive system dysfunction
6) central nervous system dysfunction, including:
   a. cerebral dysfunction
   b. depression
   c. anxiety
   d. possible 8th cranial nerve damage
7) probable peripheral neuropathy
8) immune system dysfunction with suppression

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis and conjunctivitis rests on symptoms of recurrent nasal congestion and on physical finding of mucous membrane irritation and symptoms of red, irritated, burning and watering eyes. Daniel Pryor relates these symptoms and conditions to the Alcolac odors and emissions.

2) Chronic chemical bronchitis with restrictive and obstructive defects and recurrent hemorrhage rests on symptoms of shortness of breath, wheeze, chest heaviness, cough productive of phlegm flecked with blood. The subdiagnosis rests also on physical finding of lung abnormalities of prolonged expiration and wheezing. The subdiagnosis rests also on the laboratory tests for FVC and $FEC_1/FVC$ which are both abnormally low. There is also decreased volume of the lung and scarring of the lungs - restrictive defects - as well as an obstructive defect, of the kind usual

with spasm or inflammation of the air passages. The evaluator attributed these conditions of disease and dysfunction as the chronic effects to the exposure to the Alcolac emissions.

The personal history given by Daniel Pryor related that he became a regular cigarette smoker at age 18 and continued until age 24 and consumed one and one-half pack per day. He discontinued the habit for ten years, but resumed once again at the same pace about four months prior to examination at CCA. Dr. Carnow ruled out the cigarette consumption as a cause of the chronic chemical bronchitis and pulmonary dysfunction subdiagnosis because Daniel Pryor was free of symptoms when he gave up smoking ten years before the complaints of wheeziness, chest heaviness and shortness of breath began two years ago - before the resumption of the cigarette habit. The productive coughs and the occasional flecks of blood in that sputum commenced about a year ago - and so also predated the resumption of smoking, six months before examination at CCA. His symptoms, rather, all commenced after the onset of the Alcolac productions and the emission of the pollutants. The temporal relationship of the symptoms, therefore, is not to cigarettes but to exposure to the chemicals. Daniel Pryor not only exhibits these many symptoms, however, but also manifests "a lot of wheezing in his lungs, which means that the air passages were swollen and narrowed, all over his lungs." The lung tests demonstrate a force vital capacity of only 71 percent which means "[h]e had trouble getting air out of his lungs." These bronchial conditions, he noted, are rather severe, and the cigarettes were "at most, a minimal aggravating factor."

3) Chronic gastritis rests on symptoms of gastrointestinal distress, nausea, rectal bleeding, anorexia and weight loss.

4) Liver dysfunction with abnormal lipid metabolism and abnormal protein metabolism rests on abnormal test results of

those liver functions. The depressed cholesterol and LDL cholesterol represent liver damage. The FT [free thyroxin] index was abnormally low and represents a dysfunction of the protein metabolism function of the liver.

5) Reproductive system dysfunction rests on the fertility analysis which was abnormal through the range of tests. The percent activity showed a 45% of the sperm active at 30 minutes [normal, 80%], and only 45% active at 60 minutes [normal, over 70%], and only 30% active at four hours [normal, over 60%]. Thus, at the end of four hours, only 30% of the sperm remained alive. The grade of motion and type of motion were also abnormal: the sperm were not moving rapidly and were not moving in the same direction. The total sperm count was very low at 29 million [normal, above 60 million]. Also, 23% of the sperm were abnormal. The result, the evaluator concluded, "represents a pretty bad profile."

6) Central nervous system dysfunction, including cerebral dysfunction, depression, anxiety, possible 8th cranial nerve damage, rests on symptoms of dizziness, decreased concentration and loss of memory, sleep disorders and headache. The subdiagnosis and their components rest also on the results of the neurobehavioral battery, the Zung anxiety scale and the Beck depression inventory. The neuropsychological examination disclosed a mild attentional deficit and depression, and raised a question as to a somatosensory deficit in the left side of the brain. The severe depression found, however, may impinge to explain that apparent dysfunction. It was unclear to the neurologist whether that manifested a problem of the peripheral nervous system [damage to the nerves out of the cord] or of the central nervous system [damage to the nerves out of the brain]. The Beck depression inventory was 21 [normal, 0 to 10]. The Zung anxiety scale result was 55 [normal, 0 to 44]. Thus, in addition to the test results

of a severe depression and abnormal anxiety, the tests demonstrated a nervous system deficit, whether as damage in the brain or in the peripheral nerves.

7) Probable peripheral neuropathy rests on symptoms of numbness in the hands and arms, tingling, and weakness in the legs and grip. The numbness has become recurrent with longer-lasting episodes.

8) Immune system dysfunction with suppression rests on the abnormal results of the immune panels administered by Bioscience laboratory in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| White Blood Cells: | abnormal depression of the T lymphocytes at 64 [normal, 70 to 84] |
| | abnormal depression of the segs |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally elevated OKT-10 [natural killer] cells |
| Hematologic system: | abnormal mean corpuscular hemoglobin |

The medical history of Daniel Pryor recorded occasional use of drugs: speed, LSD, downers, but not a present use. Those isolated occasions, in 1976, the evaluator commented, do not impinge on any of the symptoms, findings, laboratory results or subdiagnoses entered for Daniel Pryor. The drug experimentation was occasional only. A chronic drug use, he commented, could explain a liver pathology such as hepatitis, but there was no such condition found. In fact, Daniel Pryor was one of the few among the patient plaintiffs who had no condition of abnormal porphyrin metabolism. The damage found to the lungs, to the immune system and to the central nervous system, are not related or explainable by that occasional use those years ago.

PROGNOSIS: Very guarded.

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic conjunctivitis
2) chronic rhinitis and pharyngitis
3) vascular insufficiency
4) chronic gastroenteritis (possible porphyrin crisis)
5) liver dysfunction, including:
   a. abnormal lipid metabolism
   b. abnormal protein metabolism
   c. abnormal porphyrin metabolism
6) genitourinary system dysfunction, including:
   a. infection with bleeding
   b. electrolyte unbalance with hyperchloremia and hyperphosphatemia
7) central nervous system dysfunction with cerebellar, including:
   a. cerebellar damage
   b. severe depression
   c. severe anxiety
8) mild peripheral neuropathy
9) musculoskeletal dysfunction
10) immune system dysfunction
11) chronic chemical dermatitis
12) endocrine dysfunction
13) reproductive system dysfunction with dysmenorrhea and menorrhagia
14) hematologic system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic conjunctivitis rests on symptoms of red, irritated eyes with a swollen feeling following exposure to the Alcolac odors and emissions. It rests also on findings of reddened conjunctiva on physical examination.

2) Chronic rhinitis and pharyngitis rests on symptoms of nose irritation and congestion and on soreness of the throat. It rests also on the finding on physical examination of swollen and irritated mucous membrane with exudate on physical examination..

3) Vascular insufficiency rests on symptoms of chest pain and on physical findings of peripheral abnormalities of an abnormally red color of the legs [rubeosis - which signifies a circulatory deficiency], and diminished peripheral pulse at the feet.

4) Chronic gastroenteritis [possible porphyrin crises] rests on frequent episodes of vomiting accompanied by diarrhea, symptoms of indigestion and gastrointestinal distress, nausea and hemorrhoids. The subdiagnosis rests also on the abnormal porphyrin metabolism test results.

5) Liver dysfunction, including abnormal lipid metabolism, abnormal protein metabolism, abnormal porphyrin metabolism, rests on the results of the tests of those functions of the liver. Her triglycerides were abnormally high at 165 [normal, 30 to 150] in a woman of only 33 years. The abnormally-low FT [free thyroxin] index level of the thyroid signified an abnormal protein metabolism of that function of the liver. The abnormalities of the porphyrin metabolism function of the liver were also marked, with both a uroporphyria and a coproporphyrinuria. The uroporphyrins were at 33 [normal, 4 to 22] and the coproporphyrins were at 88 [normal 3 to 56].

6) Genitourinary system dysfunction, including infection with bleeding, electrolyte unbalance with hyperchloremia and hyperphosphatemia, rests on history of recurrent bladder infections and on abnormal laboratory test results. The urinalysis was very abnormal - more than 200 clumps of pus cells and blood in the urine. The chlorides and potassium were both abnormally elevated. The electrolytes tolerate only a narrow range within which the body can live, and so the body regulates the electrolytes with great care. The changes found, Dr. Carnow explained, were "very significant."

7) Central nervous system dysfunction with cerebellar, including cerebellar damage, severe depression, severe anxiety, rests on symptoms of dizziness, nervousness, sleep disorders and headache. They rest also on the findings on physical examination of cerebellar dysfunction and that of the 8th cranial nerve. The result of the 30 [normal 0 to 10] manifested an abnormal depression on the Beck inventory and the 57 [normal, 0 to 44] was an abnormal value on the Zung anxiety scale.

The medical history of Joyce Pryor records an entry by Dr. Allcorn, a local Sedalia physician, that she becomes very upset before the monthly menstrual period. The cross-examiner posed to Dr. Carnow whether the premenstrual syndrome was related to the central nervous system manifestations attributed to the Alcolac chemicals. The evaluator acknowledged that the records show a history of nervousness in the early 1970's for which she consulted a physician, and for which she was given medication. That condition nevertheless exacerbated so that by 1981 it was much worse. The condition of nervousness, in fact, became very severe before her menstrual periods, but Dr. Carnow concluded that the condition was related to her absorption of the chemicals. He suggested also that the condition of the nervousness was related to the changes in her porphyrin metabolism. Joyce Pryor was on a regimen of birth control pills, and the ingestion of estrogens exacerbates porphyria. Thus, the estrogens taken before the menstruations may have been a factor in the attendant nervousness - and intensified the intoxication from the Alcolac chemicals acting on her porphyrins.

The neuropsychological findings entered by Dr. William Wilson, neurological examiner for CCA, conclude with the summary: "The results of Mrs. Pryor's neuropsychological examination suggest a mild to moderate level of depression and distress in an individual with long-standing characterological problems." The

longstanding characterological problems alluded to a history of family abuse, precocious sexual play, early pregnancy at age 16, four marriages, and a pattern of possessiveness and demands for attention from the husband. The records note also episodes of drug use fifteen years before, which included "uppers, downers and heroin," and the indifference of the husband to that indulgence.

It was posed to Dr. Carnow on cross-examination - in light of these examination reports and incidents of prior history - to explain how he arrived at the subdiagnosis that the severe depression was a disease caused by the Alcolac chemicals. Dr. Carnow responded that he relied on the finding of Dr. Wilson of a mild to moderate level of depression, that the occasional drug use was fifteen years before, that the drug episode was prompted by marital discord with her then husband, that she thereafter undertook to repair her life through church activity, but that the chronic exposure to the Alcolac chemicals over the years, superimposed on her tendency to nervousness and depression, made her become progressively more irritable so that by 1984, after a period of domestic discord [this time with husband Daniel Pryor] that marriage ended in dissolution. Dr. Carnow acknowledged that the "characterological disorder" and the episode of drug use were "significant components in her depression." He insisted, however, that a person with her susceptibility to stress is more easily affected by a superimposed stress, such as toxic chemicals. He explained also that her condition of severe porphyria also impinges on central nervous system dysfunction. "It makes people extraordinarily irritable." In view of the "tremendous amount of evidence that she has been poisoned by the chemicals" as to other organ functions, that intoxication must be deemed to be a component of her depression - "a contributing cause, certainly not the sole cause."

8) Mild peripheral neuropathy rests on symptoms of tingling of the fingers and limbs, intermittent shooting pains and other sensory abnormalities following exposure to the Alcolac odors and emissions.

9) Musculoskeletal dysfunction rests on muscular pains, cramps and joint pains following exposure to the Alcolac odors and emissions, and on symptoms of weakness in the limbs.

10) Immune system dysfunction rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

Immune system:
White blood cells: total abnormally elevated at 12,300
T lymphocytes abnormally depressed at 68

[Midwest/Wheeler Laboratory Test Results, 1985]

Immune system:
Lymphocytes: white blood cells total abnormally elevated at 13,300
abnormal increase in OKT-10 [immature lymphocyte] cells
Hematologic system: abnormal mean corpuscular hemoglobin
abnormal mean platelet volume
abnormal shape [anisocytosis]
abnormal color [hypochromasia] from want of sufficient hemoglobin

The evaluator noted the progression in the elevation of the total white blood cells from 12,300 in 1984 to 13,300 in 1985, and recommended - as in the case of the others with found immune system abnormalities - successive, periodic tests. A continued elevated total at that range or beyond would cause concern for an eventual condition of leukemia.

11) Chronic chemical dermatitis rests on symptoms of skin rashes and lesions and progressive increase of facial blemishes and dry splotches on her arms and scalp following exposure to the Alcolac chemicals.

12) Endocrine dysfunction rests on the abnormal FT [free thyroxin] index test result.

13) Reproductive system dysfunction with dysmenorrhea and menorrhagia rests on symptoms of abnormally prolonged menstrual periods, episodes of bleeding, marked by painful discharges and issues of large blood clots.

14) Hematologic system dysfunction rests on the abnormal results of the tests of the hematologic system administered by Midwest/Wheeler.

PROGNOSIS: Very guarded to poor.

LINDA SANDERS AGE 23 LIFE EXPECTANCY 59.23 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) recurrent conjunctivitis
2) possible mild hearing loss
3) chronic rhinitis and laryngitis
4) chronic chemical bronchitis
5) liver dysfunction, including:
   a. abnormal enzyme, possible low-grade hepatitis
   b. abnormal porphyrin metabolism with porphyria
6) central nervous system dysfunction with depression
7) chronic chemical dermatitis
8) immune system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Recurrent conjunctivitis rests on symptoms of irritation and burning of the eyes following exposure to the Alcolac odors and emissions.

2) Possible mild hearing loss rests on symptoms of ear discomfort and the results of the audiometric tests which show a 30 decibel loss bilaterally at the 6000 cycle frequency.

3) Chronic rhinitis and laryngitis rests on symptoms of nose and throat irritation, burning in the nose and throat and recurrent infections of the throat.

4) Chronic chemical bronchitis rests on symptoms of shortness of breath, cough and chest heaviness.

5) Liver dysfunction, including abnormal enzyme, possible low-grade hepatitis, abnormal porphyrin metabolism with porphyria rests on the results of the laboratory tests of those liver functions. The heptacarboxylic porphyrins were depressed and the coproporphyrins were elevated, both abnormalities. The alkaline phophatase enzyme was abnormally elevated. That may represent a problem with the liver - a low-grade hepatitis. The globulins were also depressed. She was prescribed drugs to prevent porphyrin crisis.

6) Central nervous system dysfunction with depression rests on symptoms of depression and headache and on the abnormal Beck depression inventory, a 13 on a scale of 0 to 10. The neuropsychological findings conclude, in summary, that the examination results "are within normal limits with the exception of a mild depression whose clinical significance is uncertain given the recent birth of her child." Dr. Carnow explained that the comments of the neurologist notwithstanding, the examiner was not a clinician and the history does not suggest that Linda Sanders suffered any post-partum depression. The depression, rather, she related to the Alcolac fumes and odors. In view of her youth, he commented, there was no other explainable basis for her depression.

7) Chronic chemical dermatitis rests on symptoms of skin rash following exposure to the Alcolac emissions in 1978. Her prior history notes an episode of allergic dermatitis to detergents at her work. In April of 1977, one episode of rash over her entire body was diagnosed as viral dermatitis. Another entry for that month also notes a rash over the entire body. In November of 1978, she suffered another rash over the body, and the record

notes "soap at work." It was diagnosed by one physician as scabies and by another as contact dermatitis. There was no diagnosis made, however, that the cause was "soap at work." The rash was recurrent. Dr. Carnow entered the diagnosis, on the basis of the history of exposure to the Alcolac chemicals and on the medical history, as a chronic chemical dermatitis.

8) Immune system dysfunction rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/-Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormally depressed gamma M at 75 [normal, 80 to 310] |
| | abnormally depressed globulins |
| White blood cells: | abnormally elevated total lymphocytes at 43 [normal, 20 to 40] |
| | abnormally depressed eosinophils at 0 |
| | abnormally elevated monocytes |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally elevated OKT-10 [immature lymphocyte] cells |

The evaluator concluded that the test results show multiple derangement in various areas of the immune system. Linda Sanders married in 1980 and moved to Texas. Thus, her exposure to the Alcolac chemicals was for a period of one year and nine months. After marriage, she gave birth to three children, all of them healthy. It was the opinion of Dr. Carnow that the state of the health of the children notwithstanding, Linda Sanders, because of her youth "may have more problems than other people." To cross-examination, Dr. Carnow responded that recurrent infections were not necessarily the eventual consequences of an immune system dysfunction as she manifests. Recurrent infection is the consequence of B cell abnormality, since the B system is that arm of

the immune mechanism which relates to infections. Her abnormality, as with most of the Alcolac plaintiffs, was to the T cells -and they tend to relate "to very specific types of infections, like tuberculosis and things like that, and they relate more to destroying cancer cells."

PROGNOSIS: Very guarded to poor.

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ARNOLD SOMMERS AGE 42 LIFE EXPECTANCY 36.02 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic conjunctivitis
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic rhinitis and pharyngitis with nasal hemorrhage
4) chronic chemical bronchitis
5) acute recurrent gastritis [possible porphyrin crisis]
6) liver dysfunction, including:
   a. severe chemical hepatitis
   b. abnormal lipid metabolism
   c. abnormal porphyrin metabolism
7) genitourinary tract dysfunction with probable renal insufficiency
8) central nervous system dysfunction, including:
   a. cerebral damage
   b. probable cerebellar damage
9) peripheral polyneuropathy
10) musculoskeletal dysfunction
11) immune system dysfunction
12) hematologic system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic conjunctivitis rests on symptoms of recurrent eye irritation, itchiness, matting, watering and redness following exposure to the Alcolac odors and emissions. It rests also on finding injected conjunctiva on physical examination.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on audiometric test results of "classic neurosensory loss." On the right there was a 45 decibel loss at 4000

frequencies and a 55 decibel loss at 6000 frequencies. On the left there was a 45 decibel loss at 3000 frequencies, a 45 decibel loss at 4000 frequencies and a 60 decibel loss at 6000 frequencies.

3) Chronic rhinitis and pharyngitis with nasal hemorrhage rests on symptoms of nasal irritation with gushing nosebleeds and irritation and soreness of throat following exposure to the Alcolac odors and emissions. They rest also on physical findings of reddened throat and reddened and swollen mucosa in the nose.

4) Chronic chemical bronchitis rests on symptoms of shortness of breath and productive cough continuously during the past months. The examiner discounted a chest x-ray finding of granuloma - a residue of a primary tuberculosis endemic to those in this area of the country. It represents a tiny calcification end-result of the healing process. In this case it is "ancient" and of no diagnostic significance. Arnold Sommers was a smoker for 28 years, but denied any symptoms until exposure to the Alcolac emissions both from the plant and from the plant run-off into Little Shaver Creek which ran behind his home. The odors from these sources induce his coughs, which have become increasingly frequent and severe. This temporal relationship of the symptoms to the exposure events render the inhalations of these pollutants "the overwhelming cause of his bronchitis," Dr. Carnow concluded.

5) Acute recurrent gastritis [possible porphyria crises] rests on symptoms of nausea, from the Alcolac odors. These effects last for two to three days and then diminish until the next exposure. The medical history shows a diagnosed ulcer of the pylorus in 1976. That condition reacts to the chemical inhalations and he takes medication for the discomfort. It was the opinion of Dr. Carnow that the underlying ulcer may have healed, but in any event it was symptomatic only after exposure to the

chemicals. When the exposure ceases the symptoms also cease, so even if "some low grade underlying ulcer exists, the symptoms are of a recurrent gastritis." Dr. Carnow noted also that the laboratory test results show porphyria and the symptoms Sommers manifests of severe gastrointestinal pain and disturbance are those common to porphyria.

6) Liver dysfunction, including severe chemical hepatitis, abnormal lipid metabolism, abnormal porphyrin metabolism rests on the abnormal results of the tests of those liver functions. The coproporphyrins were abnormally elevated at 159 [normal, 10 to 109]. The porphobilinogen is 1.6 [normal 0 to 1.5] and the coproporphyrin-uroporphyrin index is abnormal at 6.6 times the copro to the uro. The liver enzymes, GGTP, SGOT, SPGT, alkaline phosphatase and LDH were all abnormal - a sign of severe chemical hepatitis caused by the Alcolac emissions. This pattern of enzyme abnormality shows the full effect of a chronic chemical inflammation of the liver. It is also characteristic of the true state of the others afflicted by liver dysfunction from the chronic exposure to the Alcolac chemicals, even though they did not all manifest the full spectrum of enzyme test abnormalities. Dr. Carnow explained that such is the nature of the enzyme system: "Certain enzymes go up and they go down, and then they may go up again, and then they go into a chronic damage phase, so it depends on when you catch them. [Arnold Sommers] shows evidence of acute damage, and of course, at this point in time, that is a serious problem." It could cause death. The lipid metabolism was also disturbed. The LDL cholesterol was high at 229 [normal upper limit, 185], the triglycerides were very high at 359 [normal upper limit, 150], and he has an abnormal phenotype.

Dr. Carnow discounted as explanation for these multiple liver functions the personal history that Arnold Sommers consumed two six-packs of beer per week. They neither acted synergistically with the Alcolac chemicals to bring about the abnormalities nor were they otherwise significant to diagnosis.

7) Genitourinary tract dysfunction with probable renal insufficiency rests on the test result of an elevated creatinine, which signifies kidney dysfunction - a matter of some concern in a person of only 42 years of age.

8) Central nervous system dysfunction, including cerebral damage, probable cerebellar damage rests on symptoms of dizziness, nervousness, tremor and headache, on physical findings of cerebellar abnormality and 8th cranial nerve damage, and on the results of the neurobehavioral battery. The neurological tests disclosed an attentional deficit - difficulty in concentration -and other abnormalities from which the neurologist derived the diagnosis: "mild diffuse brain dysfunction." Arnold Sommers described constant and intense headaches from the onset of the Alcolac operations, irritation and loss of temper. He described an episode of rage which induced him to hit his horse between the eyes with such force as to break his hand. Those responses, Dr. Carnow explained, are characteristic of chemical intoxication.

9) Peripheral neuropathy rests on symptoms of sensory abnormalities - such as tingling and numbness of the limbs and members, more pronounced on the left side. The subdiagnosis rests also on findings on physical examination of hand tremors present for more than a year, on continued headaches more severe than before, and on the decreased sensation found over the entire left leg. The subdiagnosis rests also on the results of the EMG, PNVC/motor and PNVC/sensory laboratory tests. There was abnormal

motor conduction on the lower left, abnormal sensory conduction on the lower left, and abnormal EMG on the lower left - all of which shows axonal degeneration, total destruction of those nerves.

The prior history notes a condition of polio in 1954 and vehicular accidents in 1952, when he fractured the left leg. There was a recovery from the polio, Dr. Carnow concluded, because there was no evidence of a residual paralysis and consequent atrophy of the limb. It was conceivable, Dr. Carnow explained, that the leg fracture may have injured a nerve, but given the development of numbness and tingling of the toes, hands and feet - so severe that they awaken him from sleep - it is not likely that the 1952 event is related to the conditions of peripheral neuropathy found. In any event, there is nerve damage to the right as well as to the left, so that even if the 1952 event could be said to impinge on the neuropathy to the left, it could not be related to the other damage.

10) Musculoskeletal dysfunction rests on the symptoms and findings of muscular pains and cramps and is interrelated with the symptoms, findings and laboratory results of the peripheral neuropathy manifestation.

11) Immune system dysfunction rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| White blood cells: | abnormally depressed eosinophils at 0 |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormal OKT-3 [mature T] cells<br>abnormally elevated NKH-1 [natural killer] cells |
| Hematologic system: | abnormal mean corpuscular volume<br>abnormal corpuscular hemoglobin<br>abnormal mean platelet volume<br>abnormal sizes [anisocytosis] |

12] Hematologic system dysfunction rests on the four abnormal findings in that blood system noted in the 1985 Midwest/Wheeler laboratory test results.

PROGNOSIS: Poor to very guarded [that is, closer to poor than very guarded].

JOYCE SOMMERS AGE 38 LIFE EXPECTANCY 44.60 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic conjunctivitis
2) chronic rhinitis and pharyngitis
3) liver dysfunction including:
   a. abnormal enzyme [LDH]
   b. abnormal protein metabolism
   c. abnormal porphyrin metabolism
4) genitourinary system dysfunction
5) central nervous system dysfunction including:
   a. depression
   b. anxiety
6) abnormal muscle metabolism
7) immune system dysfunction with suppression
8) recurrent chemical dermatitis

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic conjunctivitis rests on symptoms of reddened, irritated eyes and on the finding of reddened conjunctiva upon physical examination. These conditions follow upon exposure to the Alcolac odors and emissions.

2) Chronic rhinitis and pharyngitis rests on symptoms of nasal congestion, nosebleeds and soreness of the throat following exposure to the Alcolac odors and emissions. Chronic rhinitis rests also on the findings on physical examination of reddened and swollen mucosa with exudate.

3) Liver dysfunction including abnormal enzyme [LDH], abnormal protein metabolism, abnormal porphyrin metabolism rests on the results of the laboratory tests of those liver functions.

The LDH enzyme is abnormally elevated and may be evidence of inflammatory changes. The porphyrins are abnormal: the uroporphyrins are at 33 [normal, 4 to 22]; the pentacarboxylic is at 4 [normal, 0 to 3]; the coproporphyrins are at 171 [normal, 3 to 56]. This represents a gross porphyria.

4) Genitourinary system dysfunction rests on laboratory tests of abnormally elevated chlorides with pus infection.

5) Central nervous system dysfunction including depression, anxiety rests on symptoms of headache and dizziness following exposure to the Alcolac odors and emissions, from the findings on physical examination of dizziness and headaches and from the values on the Zung anxiety scale and the Beck depression inventory. The neurologist examiner found Joyce Sommers "mildly depressed and mildly anxious." The neurologist noted also a deviation of the tongue to the left, a manifestation of damage to the XIIth cranial nerve.

6) Abnormal muscle metabolism rests on symptoms of pains in the legs and thighs, knotted up legs, muscle twitches and weakness in the knees. This condition has come on within the six months prior to the August, 1984 examination at CCA.

The evaluator considered the medical history that Joyce Sommers in January of 1971 suffered injuries in a vehicular collision, among them dislocation of the hip and multi-fractures of the jaw, which confined her to a wheelchair for a time. The complaints upon which the subdiagnosis of abnormal muscle metabolism rests, however, appeared for the first time in 1984 and do not appear related to the trauma of 1971. In any event, a person with an organ already damaged is more susceptible to exacerbation from toxic chemicals. Moreover, Joyce Sommers made no complaint of the muscular disability during the interval between 1971 and 1984.

7) Immune system dysfunction with suppression rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Mitogen challenge: | abnormal PHA stimulation net CPM at 30,580 [normal, over 62,000] |
| | abnormal PHA stimulation index at 115 [normal, over 130] |
| | abnormal PWM stimulation net CPM at 6700 [normal, over 6700] |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes | abnormally elevated OKT-10 [immature] cells |
| Hematologic system: | abnormal mean platelet volume |
| | abnormal mean corpuscular hemoglobin |

8) Recurrent chemical dermatitis rests on symptoms of skin rash and itch from the Alcolac foam, odors and emissions.

PROGNOSIS: Very guarded to poor.

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JOY SOMMERS AGE 15 LIFE EXPECTANCY 67.11 YEARS

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DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic rhinitis with severe nosebleeds
2) liver dysfunction including:
   a. abnormal lipid metabolism
   b. abnormal porphyrin metabolism
3) peripheral polyneuropathy
4) immune system dysfunction
5) hematologic system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis with severe nosebleeds rests on symptoms of nose irritation and recurrent nosebleeds following intense odors and emissions from Alcolac.

2) Liver dysfunction including abnormal lipid metabolism and abnormal porphyrin metabolism rests on the results of the laboratory tests of those liver functions. The abnormal lipid metabolism manifestation rests on the very low triglycerides the test results show [16 on a normal of 30 to 150] and signifies that the liver "is not handling fats normally." That represents a dysfunction, and whether liver disease will result depends on future tests and symptoms. A finding of high triglycerides [hyperlipidemia], the witness explained, indicates a different kind of disease than low triglycerides [hypolipidemia]. The first indicates "abnormal handling of lipids," the second an "inappropriate handling of lipids."

The test results show also an abnormality in the porphyrin metabolism function of the liver: a low heptacarboxylic porphyrin. What that abnormality represents in terms of disease the evaluator could not presently say, but it was a condition found in 8 of the 31 patients examined - a "very unusual" phenomenon. It does not alter the diagnosis of toxic chemical causation because the test result was abnormally low rather than abnormally high. The abnormality of the porphyrin count - whether high or low - depends upon the stage of the porphyria and where the cascade of the stream of porphyrin metabolism is blocked. The porphyria found in Joy Sommers, the evaluator intimated, is in the incipient stage, and calls for a careful watch because of her youth - 15 years of age.

3) Peripheral polyneuropathy rests on the finding on physical examination of decreased or absent deep tendon reflexes.

4) Immune system dysfunction rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/-Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

Immune system:
Mitogen challenge: abnormal PHA stimulation net CPM

| | |
|---|---|
| | at 60,700 [normal, over 62,000] |
| White blood cells: | abnormally depressed globulins |
| | abnormally depressed eosinophil at 0 |
| | abnormally elevated total lymphocytes at 2540 [normal, 620 to 2410] |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally elevated OKT-10 [immature] cells |
| Hematologic system: | abnormal mean corpuscular hemoglobin |
| | abnormally elevated mean platelet volume |

5) Hematologic system dysfunction rests on the abnormal results of the hematologic system tests administered by Midwest/-Wheeler in 1985.

PROGNOSIS: Very guarded.

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TAMMY SOMMERS AGE 19 LIFE EXPECTANCY 63.16 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic rhinitis with nasal hemorrhage
2) chronic chemical bronchitis
3) chronic gastroenteritis [possible porphyria crisis]
4) liver dysfunction including:
   a. abnormal lipid metabolism
   b. abnormal protein metabolism
   c. abnormal porphyrin metabolism
5) probable central nervous system dysfunction with depression
6) immune system dysfunction
7) hematologic system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis with nasal hemorrhage rests on symptoms of nosebleeds and nasal congestion following exposure to the Alcolac odors and emissions. It rests also on the findings on physical examination of reddened and swollen mucosa with exudate.

2) Chronic chemical bronchitis rests on symptoms of recurrent cough productive of mucus.

3) Chronic gastroenteritis [possible porphyria crisis] rests on symptoms of diarrhea, anorexia and gastrointestinal distress and on the laboratory test results of abnormal porphyrin metabolism.

4) Liver dysfunction including abnormal lipid metabolism, abnormal protein metabolism, abnormal porphyrin metabolism rests on the results of the laboratory tests of those liver functions. Tammy Sommers at the time of examination and tests was 19 years of age, five feet three inches in height, and weighed 170 pounds. The triglycerides were elevated at 254 [30 to 154], a very high value for a young woman in the fertile period. The lipoprotein phenotype, another test for lipid metabolism, was also abnormal. These two abnormalities, when coupled together, represent a "grossly abnormal" metabolism of the lipids "almost never seen." Her condition of overweight, Dr. Carnow commented, might cause some elevation of the triglycerides, but not to the value of 254 as found. The test of the protein metabolism disclosed an abnormal alpha$_2$ globulin and signifies a dysfunction of protein metabolism. The coproporphyrins at 59 [normal, 3 to 56] represents a dysfunction of the porphyrin metabolism function of the liver and a coproporphinuria in a person so young. These findings of abnormal lipid, protein and porphyrin metabolisms, the evaluator concluded, represent "a very significant abnormality of liver function."

These multiple findings of dysfunction were explained as the action of the direct toxic agents, such as epichlorohydrin, which come to the liver to be detoxified but are so destructive that they kill liver cells. The liver [as the witness earlier explained] is the organ system with "the most responsibility for working with the P450 enzymes." These particular enzymes are manufactured in the protein strands of the liver cells called endoplasmic reticulum. When chemicals such as epichlorohydrin

[and some of the others emitted by Alcolac] "turn on this endoplasmic reticulum," they induce an abnormal production of enzymes and the derangements of the protein, lipid, iron and porphyrin metabolism systems are induced. That is because the production of these enzymes needs proteins and iron and involves the system of porphyrin metabolism. The porphyrins, the witness explained, "have a very delicate feedback system" and the unusual demand on that system engendered from the action of the Alcolac toxic chemicals on the endoplasmic reticulum causes the porphyrins to "just go out of whack." That derangement may affect the enzymes, lipids and proteins. In the case of Tammy Sommers, that derangement was extensive.

5) Probable central nervous system dysfunction with depression rests on symptoms of recurrent and prolonged headaches, on the Beck depression inventory score of "mildly depressed" and on a deficit in neurological function - episodic lapses in attention. The report noted that the depression "seems to reflect situational difficulties" - the divorce of her parents when she was young, an unhappy childhood and uncertainty as to her future. Dr. Carnow concluded that from a clinical point of view, the abnormal depression inventory at 18 [normal, 0 to 10] was significantly abnormal. The episodes of unhappy personal history, he gave as opinion, contributed to her depression, but the temporal relationship between the chemical exposures and her headaches demonstrate that the exposures were an additive to the effect of depression. The two components are not separable as causes of the manifestation of depression.

6) Immune system dysfunction rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/-Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormally depressed gamma A at 80 [normal, 90 to 340] |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally depressed OKT-11 cells at 847 [normal, 1000 to 3800]<br>abnormally elevated OKT-10 [immature] cells at 27.7 [normal, 0 to 15]<br>abnormally depressed NKH-1 [natural killer] cells at 6.7 [normal, 8 to 22] |
| Hematologic system: | abnormal mean corpuscular hemoglobin<br>abnormal mean platelet volume<br>anisocytosis<br>poikilocytosis |

7) Hematologic system dysfunction rests on the abnormal results of the hematologic system tests administered by Midwest/Wheeler in 1985.

PROGNOSIS: Very guarded to poor.

JAMES TURLEY AGE 52 LIFE EXPECTANCY 26.15 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic conjunctivitis
2) bilateral neurosensory hearing loss with 8th cranial nerve damage
3) chronic rhinitis with hemorrhage [chemical and allergic]
4) chronic pharyngitis
5) gastrointestinal dysfunction
6) cardiac dysfunction with possible early congestive heart failure
7) liver dysfunction with abnormal protein metabolism
8) genitouriary dysfunction with obstruction
9) central nervous system dysfunction with depression
10) peripheral polyneuropathy with axonal degeneration
11) musculoskeletal dysfunction with arthralgia and myalgia
12) severe immune dysfunction with suppression

The witness then explained the data on which these components of subdiagnosis rest.

1 Chronic conjunctivitis rests on symptoms of redness and irritation of the eyes following exposure to the Alcolac emissions.

2) Bilateral neurosensory hearing loss with 8th cranial nerve damage rests on the results of the audiometry tests. They show a neurosensory hearing loss on the right 55 decibels at the 4000 frequency and 85 decibels at the 6000 frequency. There was also a 40 decibel loss at the 3000 frequency, a 70 decibel loss at the 4000 frequency and a 90 decibel loss at the 6000 frequency. The hearing loss was evaluated as "moderately severe."

3) Chronic rhinitis with hemorrhage [chemical and allergic] rests on symptoms of irritated, congested nose, recurrent sneezing and clots of blood from the nose following exposure to the Alcolac odors.

The medical history of James Turley notes a fracture of the nose when a youth and a hospital episode in 1976 to reduce the blockage from that early fracture. A hospital record of April 9, 1979 indicates a complaint of chronic nasal congestion since the fracture reduction of 1976. In response to cross-examination Dr. Carnow construed the entries as an attempt to explain the cause of the continued nasal congestion. The records describe, he concluded, a severe allergic rhinitis in the past year. The manifestation of chronic rhinitis with hemorrhage entered as a subdiagnosis for James Turley, the witness explained, rests on the temporal relationship between the exposure to the odors and fumes as well as on the entry of the medical records. The effects to the nose - irritation, burning, congestion and flow of blood clots - followed upon the exposures. The medical records show that the surgery to reduce the prior nose fracture left only "a minor deviation of the septum to the right" - a condition not significant to the congestion found. The evaluator thus concluded that

the prior injury and repair may have left the nose more sensitive than would be normal, but the congestion and nasal bleeding were caused by the exposure to the Alcolac chemicals.

The witness explained the medical significance of chronic rhinitis - a condition of chronic inflammation of the mucous membrane of the nose. The membrane is a film which covers the fissures of the nose and maintains those passages clean. Thus, it functions to prevent infection. When that film becomes irritated [as from toxic agents], it dries and becomes more susceptible to infection. In cases of chronic exposure to toxic agents, the swelling of the mucous membrane becomes permanent, so that bacteria may more easily invade and infection result.

4) Chronic pharyngitis rests on symptoms of soreness of the throat following exposure to the Alcolac odors and emissions.

5) Gastrointestinal dysfunction rests on symptoms of gastrointestinal distress and constipation.

6) Cardiac dysfunction with possible early congestive heart failure rests on symptoms of arrhythmia and peripheral vascular abnormality. It rests also on the finding on physical examination of a heart "gallop."

7) Liver dysfunction with abnormal protein metabolism rests on the test results of that liver function. The total protein [electrophoresis] and total protein [SMAC] and albumin/-globulin tests were all abnormally depressed.

8) Genitourinary dysfunction with obstruction rests on symptoms of poor control and frequency of urination and nocturia. It rests also on the laboratory test result of elevated chlorides.

9) Central nervous system dysfunction with depression rests on symptoms of tremor and loss of concentration and memory of recent events. It rests also on the Beck depression inventory result of 21 on a scale of 0 to 10. That inventory, Dr. Carnow concluded, "shows a moderately severe depression."

10) Peripheral polyneuropathy with axonal degeneration rests on findings on physical examination of decreased or absent deep tendon reflexes and on the laboratory tests. The EMG, PNVC/motor and PNVC/sensory results were all abnormal. The motor nerves in the lower left extremity were abnormally slowed. The sensory conduction in that extremity was also abnormal, as was the EMG in that area.

11) Musculoskeletal dysfunction with arthralgia and myalgia rests on symptoms of crepitance, stiffness, ache, swelling and pains in the knee joints. It rests also on findings on the physical examination of pain and tenderness at the knee joints on deep palpation.

12) Severe immune dysfunction with suppression rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/Wheeler in 1985.[49]

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[49]It was the evidence that all thirty-one plaintiffs presented themselves to immune panels administered by the Bioscience Laboratory in Chicago in August of 1984 as arranged by CCA. All five of the Turley family - James, Kay, Lance, Lisa and Lyle - were included. The second immune panels were administered by KU Medical Center in December of 1984 under a protocol defined by the Alcolac experts. That protocol excluded ten of the plaintiffs - Kay Turley and Lyle Turley among them. The third regimen of immune panels were administered by the Midwest/Wheeler Laboratory in Kansas City in July of 1985 and was arranged by plaintiff immunologist Zahalsky and plaintiff expert Carnow. It included all thirty-one plaintiffs, but was administered to twenty-six of the plaintiffs at the time scheduled. Mary Landon was by that time under a regimen of cobalt for the treatment of abdominal cancer - itself a cause of immune system suppression - so an immune panel taken under those conditions would not likely yield a definitive result as to whether any dysfunction or suppression of the immune system then found was attributable to exposure to the Alcolac chemicals. The Turley family had removed from Sedalia to Texas in May of 1981 and were not available for examination at the Midwest/Wheeler Laboratory in July of 1985.

Immunologist Zahalsky was presented by the plaintiffs to evaluate the state and function of the immune system of each of the plaintiffs based upon the immune panel results then available to him - from Bioscience for all thirty-one of the plaintiffs, from K.U. Medical Center for twenty of the plaintiffs selected by the Alcolac protocol for that procedure, and from the Midwest/Wheeler panels. It was the testimony of Dr. Zahalsky that the

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormally depressed gamma A |
| Mitogen challenge: | abnormally depressed PHA stimulation net CPM |
| | abnormally depressed CON A stimulation index |
| White blood cells: | abnormally depressed total white cells |
| | abnormally depressed T lymphocytes |
| | abnormally depressed segs |
| | abnormally depressed eosinophil |

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five Turley family members did not present themselves to Midwest/Wheeler, and therefore his evaluation of the function and regulation of their immune systems rested on the Bioscience Laboratory results [which included all the Turleys] and on the K.U. Medical Center reports [which excluded Kay and Lyle of the Turley family].

The diagnoses entered by Dr. Carnow on each of the Turley family members and the manifestations of organ disease and dysfunctions of the immune system these diagnoses encompass, however, rest not only on the results of the Bioscience Laboratory immune panels, but also the results from a Midwest/Wheeler procedure.

In the case of James Turley the immune system subdiagnosis entered by Dr. Carnow was severe immune dysfunction with suppression. For Kay Turley, no immune dysfunction was diagnosed, but possible abnormal red blood cell metabolism was entered - presumably on the basis of a laboratory test of the hematologic system. For Lisa Turley, immune system dysfunction was diagnosed, once again expressly on results from both Bioscience and Midwest/Wheeler. For Lance Turley, immune system dysfunction with depression was diagnosed from the test results of those laboratories, and for Lyle Turley, immune system dysfunction with suppression was also diagnosed.

It is to be noted, that this evidence of subdiagnoses entered by Dr. Carnow for the Turley family members which rested on immune panel results from both Bioscience and Midwest/Wheeler [in apparent contradiction of the testimony of immunologist Zahalsky that the Turley family members did not present themselves for the Midwest/Wheeler regimen conducted in July of 1985] came in without objection, and hence with presumptive validity. We assume therefore, that the contradiction between the Carnow and Zahalsky testimony is only apparent. The immunologist Zahalsky gave his narrative to the court and jury on September 9, 1985. Dr. Carnow entered his immune system subdiagnoses as to the Turley family members on October 15, 1985. We assume that in that interim between the testimony by Dr. Zahalsky and Dr. Carnow, the Turley family members presented themselves in Kansas City for the immune panel regimen earlier administered to the other plaintiffs.

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally depressed OKT-11 [total] cells |
| | abnormally elevated NKH-1 [natural killer] cells |
| Hematologic system: | abnormal mean corpuscular hemoglobin |

The Turley family - James with wife Kay, and children Lisa, Lyle and Lance - were prompted by the odors, fumes and other Alcolac emissions and by the effects of those pollutants on their physical well-being, to remove to Texas at the end of May, 1981.

PROGNOSIS: Poor.

KAY TURLEY AGE 44 LIFE EXPECTANCY 38.85 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic conjunctivitis
2) chronic rhinitis and pharyngitis
3) chemical bronchitis [in remission]
4) liver dysfunction including:
   a. chemical hepatitis
   b. abnormal protein metabolism
   c. abnormal lipid metabolism
   d. abnormal iron metabolism
   e. abnormal porphyrin metabolism
5) central nervous system dysfunction with depression
6) peripheral polyneuropathy with axonal degeneration
7) possible abnormal red blood cell metabolism

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic conjunctivitis rests on symptoms of irritation, redness and itching of the eyes following exposure to the Alcolac odors and emissions.

2) Chronic rhinitis and pharyngitis rests on symptoms of nasal congestion and irritation and sore throat. It rests also on the finding on physical examination of swollen mucosa. The family moved away from the Alcolac discomfort in May of 1981, and her history notes that since that removal the irritations of the eyes,

nose and throat have not returned. The physical examination at CCA in 1984, some three years later, found her nose quite swollen. That signified, Dr. Carnow explained, that the conditions are merely in remission, but Kay Turley remains more sensitive than others to exposures of any irritant to the mucous membrane. It is the manner of chronic disease to be in occasional remission, but it never fully heals itself, the evaluator explained.

3) Chemical bronchitis [in remission] rests on symptoms of shortness of breath.

4) Liver dysfunction including chemical hepatitis, abnormal protein metabolism, abnormal lipid metabolism, abnormal iron metabolism, abnormal porphyrin metabolism, rests on the results of the laboratory tests of those liver functions. The porphyrins were abnormal: the coproporphyrins at 92 [normal, 3 to 56] signifies a coproporphyrinuria; the porphobilinogen at 1.7 [normal, 0 to 1.5] was abnormally elevated. The iron metabolism was quite low at 43 [normal, 65 to 175]. The enzyme SPGT was abnormally elevated - an indication of liver inflammation and chemical hepatitis. The lipid metabolism was also abnormal. The cholesterol was very high at 298 [normal, 140 to 270, although "you don't see a lot of women at 270"]. The LDL cholesterol was also abnormally elevated at 212 [normal, up to 185].

The danger of porphyria, the expert repeated, is that the person "can go into a porphyrin crisis at any time which can be life threatening." The disease can result in extreme agitation of the person and "very serious personality deficits," even psychotic behavior. It can manifest as very severe abdominal pain. A porphyrin crisis can also cause residual brain damage. He concluded: "There are no good treatments for this [disease]."

5) Central nervous system dysfunction with depression rests on symptoms of depression and a Beck depression inventory at 12 [normal, 0 to 10]. The subdiagnosis was "mild depression."

6) Peripheral polyneuropathy with axonal depression rests on the results of the EMG, PNVC/Motor and PNVC/Sensory test. They all show abnormal motor and sensory nerve conduction in the lower left extremities and EMG changes that represent nerve fiber destruction.

7) Possible abnormal red blood cell metabolism rests on the results of the tests administered by Midwest/Wheeler laboratory in 1985. The count of the red blood cells was depressed. The mean corpuscular hemoglobin was abnormally elevated, as was the mean platelet volume. The immune panel administered by Bioscience in 1984 disclosed two white blood cell abnormalities: somewhat elevated segs and 0 eosinophils. The immune panel administered by Midwest/Wheeler laboratory in 1985 found no irregularity in the immune system. The chart for DIAGNOSIS of Kay Turley enters no subdiagnosis of immune system dysfunction or suppression.

PROGNOSIS: Very guarded/poor.

LISA TURLEY AGE 21 LIFE EXPECTANCY 61.19 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic rhinitis [aggravated] and pharyngitis
2) chemical bronchitis
3) liver dysfunction, including:
   a. abnormal protein metabolism
   b. abnormal iron metabolism
   c. abnormal porphyrin metabolism
4) cardiac dysfunction
5) possible central nervous system damage with depression
6) peripheral polyneuropathy
7) musculoskeletal dysfunction with polyarthralgia and polymyalgia
8) probable chemical contact dermatitis
9) immune system dysfunction

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis [aggravated] and pharyngitis rests on symptoms of frequent nasal congestion and discharge and wheezing following exposure to the Alcolac odors and emissions. There was also symptom of sore throat. The subdiagnosis rests also on the finding of swollen mucosa on physical examination.

2) Chemical bronchitis rests on symptoms of cough and wheeze and upper respiratory irritation. Her episodes of cough lasted over more than a day.

3) Liver dysfunction, including abnormal protein metabolism, abnormal iron metabolism, abnormal porphyrin metabolism, rests on the results of the laboratory tests of those liver functions. The FT [free thyroxin] index was 5.7 [normal, 5.8 to 10.6] and represents an abnormality of protein metabolism. The iron metabolism was abnormal, as was the porphyrin metabolism. The uroporphyrins were abnormally elevated at 30 [normal, 4 to 22] and represented "a very serious porphyria." The coproporphyrins were 92 [normal, 3 to 56], and the porphobilinogen was elevated at 1.8 [normal, 0 to 1.5]. That condition of liver damage and dysfunction for a person that young [21 years of age] was "just very bad," the evaluator concluded.

4) Cardiac dysfunction rests on symptoms of chest pain and on the finding on physical examination of abnormal heart rhythm - a "gallop."

5) Possible central nervous system damage with depression rests on symptoms of headache, recurrent biweekly and about six hours in duration. The headaches continue to persist even though the family has been gone from the Alcolac environs in May of 1981, and are now accompanied by swelling of the head. The subdiagnosis rests also on the Beck depression inventory of 16 [normal, 0 to 10]. The neurologist examiner described her condition as that of "mild depression."

6) Peripheral neuropathy rests on symptoms of painful hands related to her joints and also on the finding of decreased or absent deep tendon reflexes on the physical examination. All of the reflexes were abnormal: at the biceps, the knee, the ankles, the triceps and wrist. The evaluator concluded: "That is evidence of a diffuse polyneuropathy."

The history of Lisa Turley shows that Lisa Turley was a competitive swimmer and incurred an injury to the shoulder during her high school attendance in Sedalia. An arthrogram was administered and she reacted to the dye. In response to the cross-examination as to whether that episode caused or contributed "to any part" of the peripheral polyneuropathy and musculoskeletal dysfunction diagnosed for Lisa Turley, the evaluator responded that the shoulder injury was the consequence of the attempt by a person with polyneuropathy and with muscles which have been metabolically poisoned "to try to use the muscles and joints the way other people do." She cannot, however, because the nerves have been damaged by the chemical poisons, and so are not attached to the joint as would be normal. In consequence, "they don't pull right," and attempted use in a normal manner - as by competitive sport - traumatizes both the muscles and the joint.

7) Musculoskeletal dysfunction with polyarthralgia and polymyalgia rests on symptoms of joint stiffness, pain and aches. The pain symptoms pervaded all of her joints and were almost continuous and unremitting. The pain persists even though the family has been removed from Sedalia since May of 1981.

8) Probable chemical contact dermatitis rests on symptoms of intermittent skin rash. The medical history contains an entry from a Texas hospital record in 1983 which diagnoses the condition as chloracne from pool chlorination. Dr. Carnow concluded that the entry was a misdiagnosis. He explained that chloracne is an acne caused by metabolic disturbance brought about

by chlorinated hydrocarbons, such as the dioxins, but not by the chlorine from chlorinated water. It is a condition that does not go away easily. The dermatitis Lisa Turley exhibits, on the other hand, is from exposure to such Alcolac chemicals as the acrylates and methacrylate. That dermatitis does not cling, as does chloracne, but comes and goes. It is not a metabolic disturbance, but a contact disturbance. The hospital diagnosis of the skin condition as chloracne was mistaken.

The evaluator discounted that her work as graphics designer was related to any of the dermatitis symptoms Lisa Turley exhibited. She gave no history of exposure to heavy solvents sometimes used in graphic design. Lisa Turley was interviewed on that aspect of her personal history by Dr. Conibear [a CCA partner], a physician with special competence in that area, and her interview discovered no such exposure.

9) Immune system dysfunction rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/-Wheeler in 1985.

[Bioscience Laboratory Test Results,1984]

| | |
|---|---|
| Immune system: | |
| Mitogen challenge: | abnormal PHA stimulation net CPM |
| White blood cells: | abnormally elevated total lymphocytes |
| | abnormally depressed total white cells |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally depressed T/4 to T/8 [suppressor to helper] ratio |
| Hematologic system: | abnormally elevated mean platelet volume |

PROGNOSIS: Very guarded to poor.

WILLIAM LANCE TURLEY AGE 22 LIFE EXPECTANCY 55.16 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic bilateral conjunctivitis
2) chronic rhinitis and pharyngitis with recurrent bleeding
3) liver dysfunction including:
   a. abnormal porphyrin metabolism
   b. abnormal prothrombin time
4) reproductive system dysfunction
5) chronic chemical dermatitis with probable hypersensitivity component
6) immune system dysfunction with depression

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic bilateral conjunctivitis rests on symptoms and physical finding of watering of the eyes following exposure to the Alcolac odors and emissions.

2) Chronic rhinitis and pharyngitis with recurrent bleeding rests on symptoms of nose irritation, congestion and discharges of blood and on the physical finding reddened mucosa on examination. He was seen by a physician in 1980 for the nasal congestion and nosebleeds, was tested for allergy, but none was found. The diagnosis was irritation. The pharyngitis component rests on symptoms of throat soreness which recur.

3) Liver dysfunction including abnormal porphyrin metabolism, abnormal prothrombin time, rests on the results of the tests of those functions of the liver. The coproporphyrin was elevated at 114 [normal, 10 to 109]. The prothrombin time was less than 60%, an abnormal value. These abnormalities both signify liver dysfunction.

4) Reproductive system dysfunction rests on the results of the fertility analysis. The total sperm count was depressed at 52,000,000. The 42% abnormal count was low. The motion and grade were also abnormal. The activity percent was also abnormally low: 60% of the sperm were still alive at 30 minutes, and 45% active at

4 hours. Dr. Carnow concluded from these results: "[H]e would have difficulty in fathering a child, but he probably could if he tried long enough."

In response to cross-examination, Dr. Carnow responded that a prior diagnosis of recurrent prostitis entered on a medical record of December 14, 1977, does not bear on the subdiagnosis of reproductive systemic function as an effect of the Alcolac chemicals. The prostate, the witness explained, has nothing to do with the production of sperm, and hence does not impinge on the reproductive dysfunction diagnosed. Rather, the prostate functions only to secrete a fluid which keeps the sperm alive until they reach the egg.

5) Chronic chemical dermatitis with probable hypersensitivity component rests on symptoms of skin rash, itch and bullae following exposure to the Alcolac odors and emissions.

6) Immune system dysfunction with depression rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | depressed gamma M at 69 [normal, 80 to 310] |
| White blood cells: | abnormally depressed total white cells |
| | abnormally depressed segs |
| | abnormally elevated total lymphocytes |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally elevated NKH-1 [natural killer] cells |
| Hematologic system: | abnormally elevated mean corpuscular hemoglobin] [two other unspecified abnormalities] |

PROGNOSIS: Very guarded.

LYLE TURLEY AGE 9 LIFE EXPECTANCY 67.81 YEARS

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) left otitis media with residual scarring
2) recurrent chemical bronchitis
3) liver dysfunction, including:
   a. abnormal protein metabolism
   b. possible abnormal porphyrin metabolism with abnormal coproporphyrin/uroporphyrin ratio
4) immune system dysfunction with suppression

The witness then explained the data on which these components of subdiagnosis rest.

1) Left otitis media with residual scarring rests on history of chronic ear infections and finding of inflammation of the middle ear on physical examination.

2) Recurrent chemical bronchitis rests on symptoms of persistent hacking cough following exposure to the Alcolac odors and emissions. It was a continuous symptom from 1978 until May of 1981, when the Turley family moved to Texas. There was an occasion of early broncho-pheumonia during the Sedalia period. That infection was cured, but the cough persisted. The treating physician ordered an immunoglobulin panel, since the recurrent and insistent coughs caused him concern that there might be something amiss with the immune system of the boy, Lyle Turley. The IgG - which protects against infection - was abnormal.

In March of 1983, two years after the move away from the Sedalia environs, Lyle Turley was diagnosed in a Texas Air Force hospital for cough and sore throat. Dr. Carnow considered that symptom a continuation of the manifestations from exposure to the Alcolac chemicals. It was merely one in a progression of infections that Lyle experienced: Infection of the eye, infection of the ear, a purulent conjunctivitis, otitis media, and the bronchial cough. Those recurrent infections were the consequence of

the abnormal immune system the laboratory tests also found - an abnormality caused by the Alcolac chemicals.

3) Liver dysfunction, including abnormal protein metabolism, possible abnormal porphyrin metabolism with abnormal coproporphyrin/uroporphyrin ratio rests on the results of the laboratory tests of those liver functions. The ratio of the copro to the uro was abnormally increased. The alkaline phosphatase enzyme was abnormally high at 240 [normal for that child 30 to 95], and may represent liver damage, particularly in view of the calcium abnormality disclosed by the genitourinary laboratory tests. The prothrombin time and lipid metabolism tests of the liver function were not administered, because of the inability to extract a sufficient quantity of blood from the youth for that purpose.

4) Immune system dysfunction with suppression rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Mitogen challenge: | abnormally depressed CON A stimulation index |
| White blood cells: | abnormally elevated total lymphocytes |
| | abnormally decreased T lymphocytes |
| | abnormally decreased segs |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune System: | |
| Lymphocytes: | abnormally elevated OKT-10 [immature] cells |
| | abnormally elevated OKT-3 [mature T] cells |
| | abnormally depressed OKT-11 [total T] cells |

PROGNOSIS: Very guarded.

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic rhinitis
2) chronic obstructive pulmonary disease
3) cardiac dysfunction with coronary artery insufficiency
4) liver dysfunction, including:
   a. abnormal protein metabolism
   b. abnormal porphyrin metabolism
5) reproductive system dysfunction
6) immune system dysfunction with suppression

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis rests on symptoms of nasal congestion and on findings of reddened mucosa and exudate on physical examination.

The medical history discloses that Ralph Winters has experienced a sinus problem intermittently the past ten years with a residual mucosal thickening. That, however, does not relate to the rhinitis attributed to the Alcolac chemicals - which relates to the tissues of the nose - Dr. Carnow explained, but to the tissues around the opening of the sinus.

2) Chronic obstructive pulmonary disease rests on symptoms of cough and on the abnormal results of the $FEV_1/FVC$ pulmonary test. He had the ability to get only 58% of the air out of his lungs in one second, rather than the normal 70% to 75%. That was evidence of a moderate obstructive defect.

3) Cardiac dysfunction with coronary artery insufficiency rests on the EKG. It shows that the heart is not getting enough blood, a condition diagnosed as coronary insufficiency.

4) Liver dysfunction, including abnormal protein metabolism, abnormal porphyrin metabolism, rests on the results of the

tests for those liver functions. There were three abnormal porphyrins: the pentacarboxylic was elevated at 5 [normal, 0 to 4]; the coproporphyrin was elevated at 131 [normal, 10 to 109]; the porphobilinogen was elevated at 1.9 [normal, up to 1.5]. The protein metabolism was also abnormal: the gamma globulin A was elevated.

5) Reproductive system dysfunction rests on the fertility analysis. The total sperm count was normal, but the activity was abnormal [only 60% live sperm after 30 minutes [normal, greater than 80%], and there was an abnormal morphology [40% of the live sperm was abnormal].

6) Immune system dysfunction with suppression rests on the immune panels administered by Bioscience in 1984 and Midwest/-Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormal elevation of gamma G at 1510 [normal, 700 to 1500] |
| Mitogen challenge: | abnormal PHA stimulation net CPM at 24,555 [normal, over 62,000] |
| | abnormal PHA stimulation index at 89 [normal, over 130] |
| | abnormal CON A stimulation net CPM at 4800 [normal, over 12,000] |
| | abnormal CON A stimulation index at 18 [normal, over 40] |
| | abnormal PWM stimulation net CPM at 6994 [normal, over 8000] |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | abnormally elevated NKH-1 [natural killer] cells at 29.4% [normal, 8 to 22%] |
| | abnormally elevated total NKH-1 [natural killer] cells |
| Hematologic system: | abnormal mean corpuscular hemoglobin |
| | abnormal mean platelet volume |
| | abnormal random distribution |

PROGNOSIS: Very guarded to poor.

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) chronic rhinitis
2) early vascular insufficiency
3) liver dysfunction, including:
   a. abnormal porphyrin metabolism
   b. abnormal lipid metabolism [aggravated]
4) central nervous system damage, including:
   a. cerebellar dysfunction
5) mild peripheral neuropathy
6) immune system dysfunction with suppression
7) polyarthralgia and polymyalgia

The witness then explained the data on which these components of subdiagnosis rest.

1) Chronic rhinitis rests on symptoms of nasal congestion and sneezing following exposure to the Alcolac odors and emissions and on the findings of reddened mucosa and exudate on physical examination.

2) Early vascular insufficiency rests on the finding on physical examination of diminished peripheral pulse and on the abnormal triglycerides result of the protein metabolism test. To cross-examination inquiry whether the diagnosis of diabetes in 1980 caused or contributed to cause the early vascular insufficiency manifestation, Dr. Carnow responded that it did not. He explained that the modus operandi of chemicals and of the diabetes condition differ as a cause of vascular insufficiency. The chemicals work through the nervous system to narrow the blood vessels to bring on that condition. The physical examination of her peripheral nervous system disclosed diminished pulses in the lower extremities of Genevieve Withers. The diabetes, on the other hand, "work[s] through a slow process of hardening of the artery and laying down of cholesterol and narrowing the artery [which]

would happen over many years." The diabetes of 1980 could not have caused the early vascular insufficiency, but it is possible it could have contributed.

3) Liver dysfunction, including abnormal porphyrin metabolism, abnormal lipid metabolism [aggravated] rests on the results of the laboratory tests of those liver functions. The heptacarboxylic porphyrin was depressed. The lipid metabolism was extremely disturbed: the triglycerides were very high at 600 [normal, to 150], which indicated that "[h]er blood vessels were being coated with cholesterol." The phenotype was also abnormal. Dr. Carnow described the condition as "very dangerous."

In response to cross-examination whether the 1980 condition of diabetes caused or contributed to cause the abnormal lipid metabolism manifestation, Dr. Carnow responded that "her lipids were probably aggravated by the development of the diabetes." He noted a prior history of occasional episodes of high blood sugar in 1966 and 1968. Also, in 1976, her triglycerides were at about 440, already considerably above normal. Her uric acid was also elevated. There was never any sugar in the urine until 1980, however, and the earlier occasions of elevated sugar were not diabetes. Thus, there was a tendency to diabetes, but the condition itself did not appear until 1980. Her lipids may have been aggravated by the diabetes, but at 600 they were even much higher than one would expect in a diabetic.

4) Central nervous system damage, including cerebellar function, possible cerebral dysfunction, rests on symptoms of decreased concentration and recent memory loss, sleep disorders and headache, and on the finding of swaying on physical examination.

5) Mild peripheral neuropathy rests on symptoms of cramps, pain, tingling and numbness in the hands and feet.

6) Immune system dysfunction with suppression rests on the results of the immune panels administered by Bioscience in 1984 and Midwest/Wheeler in 1985.

[Bioscience Laboratory Test Results, 1984]

| | |
|---|---|
| Immune system: | |
| Immunoglobulin: | abnormally elevated gamma A |
| | abnormally decreased gamma M |
| White blood cells | abnormally elevated total lymphocytes |
| | abnormally elevated T lymphocytes |
| | abnormally depressed segs |

[Midwest/Wheeler Laboratory Test Results, 1985]

| | |
|---|---|
| Immune system: | |
| Lymphocytes: | depressed NKH-1 [natural killer] cells |
| Hematologic system: | abnormal mean corpuscular hemoglobin |
| | abnormal random distribution |

PROGNOSIS: Very guarded to poor.

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The PROGNOSIS was the final entry on the DIAGNOSIS chart of each of the patient plaintiffs. The prognosis, as Dr. Carnow explained, constituted the forecast as to "what can this patient expect as a result of the exposure, the absorption and the damage that was done." The prognosis was determined by "what we know about what these chemicals can do" in terms of the personal profile of the patient and the extent and kind of damage already done to the person. The prognosis good means that after a brief discomfiture, the patient almost certainly will recover. The prognosis guarded means there is some irreversible damage and impairment of the quality of life, and some life shortening. It means the condition will probably not worsen much. Very guarded means that the person has been damaged, there is life shortening and the condition will deteriorate over time. It is characteristic of chronic disease, "that people go down sort of stepwise" - the body deteriorates, attempts to recover, but is never restored to the

condition before injury. Poor means the condition is life shortening and will deteriorate rapidly, and survival over a period of time is questionable. Negative means that the person probably will not stabilize and will not survive more than months or a few years.

The prognoses actually entered as to the several plaintiffs ranged from very guarded to negative. That means that the forecast as to every plaintiff was for a life shortening and a condition of organ and function damage certain to deteriorate over time. In the case of Mary Landon, the negative prognosis forecast that she would not survive more than months or a few years. She died in May of 1986 while appeal pended. In addition to the discrete prognosis entered for each, Dr. Carnow forecast as a reasonable medical certainty that 11 of the plaintiffs are "at extraordinary risk to cancer," and attributed two reasons. First, they have been exposed over a long time to a number of Alcolac carcinogens: epichlorohydrin, glycidyl ether, and ethyl acrylate. Among them, epichlorohydrin is a direct carcinogen and needs no metabolic activation to start cancer other than the chemical itself. Epichlorohydrin also attacks the chromosomes. There are also mutagens among the Alcolac toxicants: epichlorohydrin, methylene chloride, ethyl acrylate, dimethyl sulfate. Their effect is to alter DNA and the way a cell reproduces itself, and so may induce a line of deformed and even neoplastic cells. Second, the immune system, the major defense against cancer, has been severely damaged in most plaintiffs. The effect of chronic exposure to epichlorohydrin, dimethyl sulfate and toluene is to toxify the cells and suppress the immune system. Thus, these same chemicals which alter DNA also alter the immune system so that they cannot fight the cell pathology engendered by the chemical alteration of DNA. It was the opinion of immunologist Zahalsky that all thirty-one plaintiffs suffer from "systematic, progressive chemical

intoxication" of the immune system and are subject increasingly to a variety of diseases, "including, but not exclusively, neoplastic disease [cancer]."

The forecast of an increased risk of cancer was articulated by Dr. Carnow in these terms: "[I]f these people live long enough, they will die from a malignancy." That prognosis, he explained, was qualified by a competing risk. Since the plaintiffs in one degree or another all suffer from systemic poisoning, and since the latency period for cancer extends to some twenty years, it may well be that some of them may die from another organ disease caused by the exposure to the Alcolac chemicals - such as porphyria or liver disease - before the cancer. The prognosis of increased risk of cancer rested on a reasonable medical certainty, and could not be quantified further. He explained: "There is not enough information in science to do that."

The environmental medicine expert came to the determination that the manifestations diagnosed as chronic system intoxication were the result of exposure to the Alcolac toxicants only after other alternative causes of the source of injury and of the biological disease found were eliminated.

That expert opinion was a conclusion drawn from these assumed facts already in evidence: A cluster of plants operated in the industrial area a short distance southwest of Alcolac - DeLong was a welding operation, Quality Fiberglass manufactured TV satellite dishes, Missouri Pressed Metals manufactured bearings and bushings, and the Missouri Pacific Railroad yards maintained railroad equipment. Two of these plants, Missouri Pressed Metals and Missouri Pacific Railroad, used chemicals of some toxicity in their operations.

The evidence was that Missouri Pressed Metals commenced manufacture several years before Alcolac did in 1978. In the production process, Missouri Pressed Metals used a solution of

trichloroethane mixed with animal fat to remove grease from the bearings and bushings. Trichloroethane evaporates very quickly and gives off the odor of alcohol. It has a very low toxicity but causes irritation to the eyes and irritation to the skin - symptoms many of the plaintiffs exhibited. The trichloroethane vapors were vented into the air during the dipping process. The sludge residue of the dipping mixture was accumulated in a 55 gallon drum and took about one year to fill. It was formerly spread around the plant acreage as a weed-killer for disposal. It is now transported away. The advisory data from Dow Chemical Company, the manufacturer, states that small amounts of the chemical can be placed on the ground and allowed to evaporate safely. These methods of venting of the vapors and occasional disposal of the liquid residue have never been cited by OSHA, which periodically inspects the plant, or by the Missouri Department of Natural Resources. There is no evidence that anyone has ever experienced any alcohol odor from any source, Missouri Pressed Metals included.

There was evidence that the Missouri Pacific Railroad car repair yard was registered with the EPA as a hazardous waste generator in 1980. That facility operates not only a repair shop but a sign shop. The signs are made by a silk screen process which uses a Dupont thinner composed of 66 percent toluene, among other chemical compounds. The spray paint guns used in the car repair shop are cleaned with the same Dupont thinner used in the sign silk screen process. The thinner was identified as a potentially hazardous waste by the DNR, "probably toxic due to its toluene content." The thinner waste liquid is generated at the rate of about 100 gallons per month and has been disposed of on-site for at least eight or nine years in a series of pits on the edge of the property. A transposition of gallons into pounds

of toluene of 100 gallons of liquid 66% toluene equals 66 gallons of toluene. That reduced to 528 pounds per month, or 6336 pounds per year, or about one-third of the annual use by Alcolac.

It was the virtual unanimous evidence from the plaintiff and nonplaintiff residents of the environs that there was no annoyance from the odors or emissions from these and the other industrial sources, but only from Alcolac. Some among the witnesses described an odor of lacquer from Quality Fiberglass on occasion, but fleeting and without physical consequences. A number of the witnesses resided close by these plants for some years, but they were never the cause of annoyance, complaint or symptom. The public official witnesses agreed that there had been no citizen complaints of odors or emissions or physical complaints from the operation of the other plants in the vicinity - but only as to Alcolac. It was not until Alcolac began operations in May of 1978 that any odors or other emissions were palpable, and not until then were any ill effects to their persons felt. This, the environmental medicine expert Carnow testified, was already known to him from the interviews of the patient plaintiffs.

These facts assumed, Dr. Carnow gave the opinion that none of the manifestations of the diagnoses of the plaintiffs could be attributable to any of these industrial sources as the cause of their bodily diseases, except only Alcolac. In summary, the determination by the expert that Alcolac only was the point source for the bodily manifestations of disease diagnosed in every plaintiff derived from (1) the effect of the evidence that those who lived in the environs, plaintiff and nonplaintiff alike, identified Alcolac as the source of the odors and emissions which caused them symptoms and discomfort (2) on the miniscule quantity of toxic matter generated by Missouri Pressed Metals and Missouri Pacific Railroad compared with the hundreds of thousands of pounds of epichlorohydrin and other toxic compounds used by Alcolac every

year (3) on the systems used by the other industrial operations to dispose of their toxic waste - Missouri Pacific deposited the toulene mixture into covered pits - and Missouri Pressed Metals vented them in a manner not objectionable to the DNR (4) on the "tremendous amount" of toxic pollutants emitted into the atmosphere from thousands of pyrolysis products generated from the malfunctioning liquid incinerator and from the regular discharge of monomer waste, still toxic and contaminated, into the bioponds, and (5) on the known effects of many of these toxic wastes emitted by Alcolac as metabolic poisons to the entire body system.

The determination by the environmental medicine expert that the bodily diseases found in each plaintiff were the result of exposure to the Alcolac toxic emissions, in turn, was through the process of differential diagnosis. It is a diagnosis of disease derived only after other recognized alternative causes have been eliminated. In that exercise, Dr. Carnow took into account the personal and medical history of each of the patient plaintiffs, the impingement of the work occupations, the age, gender and other idiosyncratic factors. Thus, a preexistent diabetes impinges on the protein metabolism function of the liver - and was taken into account to eliminate the Alcolac chemicals as the cause of a manifestation of disease based on an abnormal test result of that function. Thus, also, a symptom of blurred vision - a common complaint among all the plaintiffs - can be caused by cataract as well as by a swollen cornea with iritis, a condition expected from exposure to a number of the Alcolac chemicals. The manifestations as finally entered on the DIAGNOSIS chart of each plaintiff was as finally defined by the interview history of each, the findings on physical examination, the results of the laboratory tests, the testimony of the plaintiff at the trial, and reexamined by the testimony and cross-examination of Dr. Carnow. The manifestations were then expressed as caused by the Alcolac

toxic emissions, aggravated by them, or in some cases, reconsidered and deleted from diagnosis. The diagnosis itself was in each case the generic determination: chronic systemic chemical intoxication caused by the Alcolac toxic chemicals. There is, however, no differential diagnosis to explain the chemical systemic intoxication except "another systemic poison" - and there was no evidence that "all of these people" had been poisoned in some other way. Environmental medicine, he explained, deals with probability, and in circumstances such as plaintiffs present of common symptoms, physical findings and abnormal test results as to multiple organ systems, the cause of the systemic chemical intoxication was Alcolac.

B
The Medical Experts
and
Biological Causation
For the Defendants

It was the role of Dr. Edward A. Emmett and Dr. R. Kerby to derive opinion whether the toxic emissions from Alcolac were the cause in fact of the symptoms, biological manifestations and end points of disease of the plaintiffs, as diagnosed by Dr. Carnow.

In May of 1984, Alcolac undertook to consult with Dr. Greenberger, Chairman of the Department of Medicine at the University of Kansas Medical Center, to arrange for the interviews and physical examination of the thirty-one plaintiffs. Dr. Greenberger was preoccupied with regular duty and referred the task to Dr. Kerby, a physician, board certified in internal medicine and specialist in pulmonary disease.[50] To prepare for the interview

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[50]Dr. Kerby then served as Professor of Medicine in the Division of Pulmonary Disease and Critical Care Medicine at the University of Kansas Medical Center. He pursues academic medicine which involves teaching through the subspecialty level as a clinician who treats patients as an instructional device. He also treats patients privately. He has participated in epidemiological projects on tuberculosis and studied the interrelationship of

and examination procedures, Dr. Kerby had access to the Carnow, Conibear & Associates records of the examinations, symptoms, laboratory results, prior medical history and treatment and diagnostic conclusions entered by Dr. Carnow as to each of the thirty-one plaintiffs. It was the purpose to consult a specialist in pulmonary disease for these procedures because of the pulmonary aspects of the complaints and diagnoses entered by Dr. Carnow. It soon became apparent "that it involved multi systems," and hence the examinations were organized to that end.

Dr. Edward A. Emmett,[51] a physician in internal medicine and occupational medicine, and a board certified toxicologist, was engaged by Alcolac to consult with Dr. Kerby and to adopt a protocol for the examinations and laboratory tests to be administered to the thirty-one plaintiffs. The responsibility for the physical examinations of the plaintiffs was delegated to Dr. Kerby, and the selection of the laboratory tests to be administered was settled by Dr. Kerby and Dr. Emmett. The protocol of the laboratory procedures was organized only after consultation

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immunology and pulmonary disease with Dr. Stechschulte, immunologist witness for Alcolac.

[51]Dr. Emmett serves as Director of the Division of Occupational Medicine of the School of Hygiene and Public Health at Johns Hopkins. He is board certified in toxicology in the United States and board certified in occupational medicine in Australia. Environmental medicine, he explained, is a counterpart of occupational medicine and is now subsumed within that specialty. He sees patients regularly for occupational exposures and illnesses and specializes in occupational dermatology. Among his extensive professional credits, is affiliation by the Division of Occupational Medicine with the World Health Organization [WHO] - the branch of the United Nations which deals with the subject of health - and participation in the working groups of the International Agency for Research on Cancer [IARC], which deals with substances suspected as carcinogens. The working groups determine from the evidence whether the suspect substance is a cancer agent in humans or animals. The findings are then distributed for comment to peer members of IARC throughout the world. Among his many other credits are roles in numerous epidemiological studies.

with the staff at the University of Kansas Medical Center expert in the diagnosis and treatment of the various organ systems the records furnished by the plaintiffs disclosed were affected by exposure to the Alcolac chemicals. There was no reason to question the accuracy of the analyses of the biochemical tests done by Bioscience Laboratory [a facility the University of Kansas Medical Center often used for special tests] witness Kerby explained, and hence it was decided that those test procedures would not all be duplicated.

Tests conducted at other facilities were also not all duplicated since those values were also generally reasonable and new tests were not necessary to valid diagnosis. The fertility analysis was not repeated, nor the audiometry, nor the neurobehavioral battery, nor the porphyrin fractions. The other procedures - such as the neurophysiological examination, pulmonary function test and thyroid panel - were administered selectively. The prothrombin time - a test of ability of the liver to process a vitamin which affects the coagulation of the blood - was repeated as to each plaintiff. The reason was that the results of the tests administered under the auspices of CCA all reported the same abnormality. In fact, however [as Dr. Emmett acknowledged], that very anomaly - that they "looked funny" - was noticed by Dr. Carnow, who caused them to be rerun. The retest noted two affirmative prothrombin time results - for Amber Cross and Lance Turley. Dr. Emmett did not recall the results of the retests.

The immune panels were delegated to clinical immunologist Stechschulte [although actually administered by Dr. Bodensteiner of the department of immunology] with the prerogative to "perform whatever immunologic tests he saw fit." That phase of the K.U. protocol excluded ten of the plaintiffs so that only twenty-one immune panels were administered. The selection was prompted by consideration of the immune deficiency states already

diagnosed by the plaintiff experts as well as the need in those cases "to be more certain there was or was not the presence of some defect in the immune system."

Dr. Emmett was asked to define a reference range and its role in the evaluation of diagnostic tests. A reference range, he explained, is "what you would expect most people to have in some kind of reference population - [usually] ninety-five percent of the [population]." Dr. Kerby concurred that a test value outside of the usual range - where 95% of a given population would test - is considered an abnormal test value. A laboratory response, however, may vary from day to day for numerous reasons. Thus, in terms of diagnosis, a value outside the reference range of a diagnostic test must be interpreted in the context of history, examination, other tests and other relevant medical considerations. The witness Emmett acknowledged that he could not recall ever having seen results of the particular immune panel tests without accompanying reference ranges, as was the case with the K.U. laboratory workup.

Among the organs tested for disease and dysfunction by Bioscience for the plaintiffs and by K.U. Medical Center for Alcolac was the liver. The toxicologists agreed that the liver was a target organ of allyl alcohol, glycidyl ether, toluene, and virtually all of the other chemicals used and identified as part of the Alcolac monomer process. The liver, internist Kerby explained, has many multiple functions. It metabolizes, excretes metabolic waste and synthesizes substances important to the bodily function. There are standard tests to determine the integrity of the liver cell and these functions. The test for the integrity of the liver cell involves a group of enzymes - SGOT, SGPT and LDH - normally within the cell, but which leak into the blood when a liver cell is damaged. The detection of these substances in the blood in increased quantities signifies damage to the liver. Toxic

chemicals - such as the components of the Alcolac monomers - kill liver cells and so increase the incidence of these substances in the blood. The liver also excretes the metabolic waste out of the blood. The bilrubin, a breakdown product of hemoglobin, and the enzymes GGPT and alkaline phosphatase are present in the blood when the excretory function of the liver is impaired. The liver also synthesizes substances that are important to the bodily system. The clotting of the blood is an aspect of liver synthesis function and is measured by the prothrombin time test. Other substances commonly measured to test the synthesis function of the liver are the serum albumin and cholesterol - a lipid which increases in the blood when the synthetic function of the liver is diseased.

The tests described by Dr. Kerby for the integrity of the liver cells and liver function were all administered to all of the plaintiffs by Bioscience under the CCA procedure. The tests [except for the prothrombin time] were done by K.U. Medical Center only very selectively among the several plaintiffs. The results of the liver function panels returned by Bioscience show at least two abnormal test values for every plaintiff. A subdiagnosis entered by Dr. Carnow as to every plaintiff was liver dysfunction in some particular[s]. The experts Kerby and Emmett did not quarrel with the abnormal values reported by these panels of tests. They did not rerun them on each of the plaintiffs as a matter of routine because the Bioscience Laboratory does reputable work. They did question nevertheless, that the abnormal test values - in themselves - were clinically sufficient to sustain the diagnoses entered by Dr. Carnow.

A recurrent subdiagnosis entered by Dr. Carnow, and noticed by these Alcolac experts, was abnormality of the porphyrin metabolism function of the liver. The porphyrins, these experts explained, are necessary for biological reactions in all the cells

in the body. Therefore, the ability to synthesize porphyrins is synonymous with life. The hemoglobin of the red blood cells - which binds the oxygen transported into the bodily tissues - are produced in part from porphyrins. The porphyrins themselves are the end result of a series of syntheses from chemical delta ALA through intermediate steps [described by Dr. Carnow as "cascades"] to the porphobilinogen porphyrin, to the uroporphyrin, then to the carboxylics, then to coproporphyrin, then to protoporphyrin. This process of porphyrin synthesis is catalyzed by a series of enzymes along the pathway [or "cascade"].

The diseases associated with porphyrin metabolism, Dr. Kerby explained, result when a step in the process of porphyrin synthesis is blocked. That may occur from a deficiency in one of the enzymes that catalyze these steps. When the next step of porphyrin synthesis is blocked, the porphyrins accumulate into an unhealthy state. The vast number of enzymatic defects, as well as other forms of porphyrin disease, are inherited. The most significant disease of porphyrin metabolism is porphyria. It results from a block between the porphobilinogens and the uroporphyrins. These compounds accumulate and are toxic to the nervous system. They cause destruction of the peripheral nerves, spinal cord processes and brain. The symptoms of a block there are pain in the belly [because those nerves are involved], paralysis, peripheral nerve dysfunction, numbness and tingling. Another block which causes porphyria is at the uroporphyrin to coproporphyrin step. The uroporphyrin accumulates in the blood and gets into the tissues. The result is skin photosensitivity - rashes, boils, blisters, scars, and skin disfigurement to the part of the body exposed to sunlight. That disease is known as porphyria cutanea tarda.

Toxic chemicals may also cause porphyria. The dysfunction caused by toxic chemicals is usually a block at the uroporphyrin to coproporphyrin step. The porphyrin which accumulates in the urine as a consequence is most commonly the uroporphyrin. The result is the same skin photosensitivity and porphyria cutanea tarda as from a block at that step of porphyrin synthesis from genetic causes. To test for disease the quantity and fractions of the porphyrins accumulated from the dysfunction of metabolism are measured in the urine of the person. That was the test administered to all of the thirty-one plaintiffs at the Mayo Clinic by the HPLC technique. The quantitation and fraction of the porphyrins was a test the K.U. protocol excluded altogether.

Dr. Kerby deemed it important to separate two phenomena - porphyrinuria and porphyria. Porphyrinuria is the presence in the urine of porphyrins, usually coproporphyrin or uroporphyrin. It is a not uncommon condition. Porphyria is a disease associated with the substances in the body cells. It is "really a very rare disease," Dr. Kerby explained. The presence of mildly elevated levels of porphyrins of various types in the urine - as the review of the Mayo Clinic tests disclosed - he concluded, does not signify an abnormal state. Nor is there any "scientific credibility" that "too few porphyrins" represents an abnormal disease state - as the diagnosis by Dr. Carnow assumes. He gave opinion that "a persistent problem with porphyrin metabolism related to environmental exposures to a chemical would probably produce a consistent defect." It would be expected - since the block in such cases is between the uroporphyrins and the coproporphyrins - that the porphyrin to appear in the urine be uroporphyrin. The porphyrin measurements in the urine of the plaintiffs, however, show a variety of patterns - only four among them with elevated uroporphyrins. To signify porphyria, however, the expected uroporphyrin elevation is many times the normal level. It

was the conclusion of Dr. Emmett - whose expert testimony of the subject was congruent with that of Dr. Kerby - that from analysis of the Mayo Clinic test results, none of the plaintiffs had the porphyria disease. Dr. Kerby acknowledged, nevertheless, that the eighty-seven percent incidence of porphyrin test abnormalities in a population of thirty-one [plaintiffs] was "[p]robably a bit high - probably abnormal." He acknowledged also that the state of abnormality could be caused by toxic chemicals. The opinion of toxicology expert Emmett intimated that epichlorohydrin can cause porphyria. Another toxicology expert for Alcolac, Dr. Schwartz, deemed it "certainly plausible" that exposure to that compound can cause porphyria since epichlorohydrin has a "fairly extensive ability to interact with the liver."

Another test procedure administered to all of the plaintiffs by CCA was the audiometer. The experts Kerby and Emmett did not include that test in the K.U. protocol but relied on the results of the procedure conducted at the CCA facility to derive diagnosis of hearing loss. To evaluate hearing loss, Dr. Carnow applied the results of the audiometric tests to the Spoor Graphs - composites of the average hearing levels at different frequencies for persons of each age and gender. Hearing loss - in one degree or another - from damage to the 8th cranial nerve from the Alcolac toxic chemicals was entered as a subdiagnosis as to seventeen of the thirty-one plaintiffs tested. Alcolac toxicologist Emmett acknowledged that toxic chemicals can damage the 8th cranial nerve and so cause hearing loss. The two most common causes for the condition, he explained, were age [the condition presbycusis] and occupational noise. Conductive loss - not from damage to the cranial nerve, but from an obstruction to the conduction of the sound to the nerve - also results in impairment of hearing. The review of the audiometric results from CCA and from the history and records of the plaintiffs prompted the experts Kerby and

Emmett each to attribute the hearing loss shown by the audiometer in each case either to age, occupational noise or to conductive loss.

Thus, it was the conclusion of the Alcolac medical experts that in no instance was the hearing loss tested by the audiometer induced by exposure to toxic chemicals. It was the response of toxicologist Emmett, moreover, that none of the chemicals identified as used in the monomer production process affected the 8th cranial nerve or the hearing function. Emmett acknowledged on cross-examination, however, that authoritative texts report high frequency hearing loss in rats exposed to toluene, a loss apparently irreversible. Those authorities also report, Dr. Emmett agreed, that the observed tone pips in the rats exposed to toluene "are similar to those seen in human patients with sensorineural hearing loss" and "are in contrast to the patterns observed in patients with conductive hearing loss." Emmett qualified that agreement with the observations that these findings were at levels "more high-pitched than tested normally on the human audiograms." He commented also that "[t]he rats were exposed to very high levels of toluene."

The protocol adopted by the Alcolac experts excluded fertility analysis of the male plaintiffs as well. The experts Kerby and Emmett resorted to the results of the sperm tests conducted at the Cook County Hospital for evaluation and diagnosis of the testicular function. Seven of the male plaintiffs presented themselves for analysis at the Cook County Hospital, and all seven of them tested abnormal in at least two categories. The test procedure measured the count of live sperm, the grade, motion, activity and morphology. The count measures the number of sperm per millimeter of semen. The grade measures the number of primitive cells, pathologic sperm. The motion measures the direction

of movement - motion in the same direction facilitates fertilization of the egg. The activity measures how many sperm remain alive at the elapse of periods of time - 60% should remain in motion at the end of 4 hours in order that the number needed to travel into the fallopian tube to fertilize the egg remain alive for the 72 hours necessary for that trip. The morphology measures the appearance of the specimen - a quantity of more than 20% poorly made sperm in one specimen is abnormal.

A single ejaculation produces 100,000,000 sperm. In his evaluations of the Cook County Hospital measurements, Dr. Carnow applied the 60,000,000 live sperm count in a specimen as the normal standard, a count of between 40 and 60 million as borderline abnormal, and a count below 20,000,000 as sterility. Dr. Emmett applied the count of 20,000,000 as the normal - at "the lower level of the reference range." That standard, Dr. Emmett explained, was the more contemporary and reflected the diminished male fertility in developed countries. He added, however, that "in the United States the average now is forty-eight million, which actually falls below the sixty unit [applied by Dr. Carnow]." He acknowledged nevertheless that the standard applied by Dr. Carnow to derive evaluations and subdiagnosis of the seven plaintiffs was the normal reference range noted in the authoritative text, Casarett and Doull's Toxicology (Second ed. 1980). That authority also reports that a sperm count of less than 20,000,000 generally constitutes sterility.

Dr. Emmett found no clinical significance to the fertility test results entered at the Cook County Hospital for the seven plaintiffs. The diagnoses entered separately by Dr. Emmett and Dr. Kerby found the reproductive systems of all of them normal. Dr. Emmett did comment that there was one among the seven who "should probably have repeated tests." He did not identify the one, nor was any test repeated. He gave opinion also that there

was no basis to conclude that any of the Alcolac chemicals cause infertility. He acknowledged that "sperm count or fertility testing [can be] a marker for chemical damage," but gave opinion that there was no basis to conclude that any of the Alcolac chemicals cause infertility. It was the opinion of Dr. Kerby, however, that plaintiff "Dan Pryor's problem [with infertility]" - which that expert attributed to alcoholic consumption, "could be caused by toluene." That compound is noted by Casarett and Doull's Toxicology (Second ed. 1980) as toxic to sperm and male reproduction.

The commonality of tingling, numbness and other unusual sensations in the limbs of the ninety persons resident in the environs of Alcolac - plaintiffs and nonplaintiffs alike - and knowledge that exposure to a number of the Alcolac chemicals induces such symptoms prompted Dr. Carnow to administer to all thirty-one plaintiffs a neurological examination and nerve conduction/electromyograph battery. The Alcolac protocol included only three of the plaintiffs for comparable neurophysiological tests at K.U.: John Phillips, Charlotte Phillips and Arnold Sommers.

It was the testimony of Dr. Emmett that numbness and tingling of the limbs is a common complaint from exposure to toxic chemicals. These symptoms suggest, among other diseases, symmetrical polyneuropathy. It manifests in symmetry because the chemicals affect the long nerves of the spinal cord on both sides. It starts in the hands or legs and moves upward. There are other causes - alcohol and diabetes among them. To reach diagnosis, tests are done of reflextion and sensation, and then studies of nerve conduction. Emmett diagnosed three among the plaintiffs - John Phillips, Charlotte Phillips and Arnold Sommers - for symmetrical polyneuropathy. Dr. Emmett and Dr. Kerby concluded that the state of disease found in all of them was not attributable to

Alcolac, but was from other cause. It was the subdiagnosis entered by Dr. Carnow that twenty among the plaintiffs exhibited a state of peripheral polyneuropathy caused by exposure to the Alcolac chemicals. Dr. Emmett was not "particularly surprised" that the tests disclosed that one-half of the plaintiffs exhibited abnormal sensory conduction in the lower extremity, one-fourth of them exhibited abnormal motor conduction in the lower extremity, or that one-fifth of them exhibited abnormal electromyographic [EMG] studies. Nor did Dr. Emmett consider significant to diagnosis the statistic simpliciter that 78% of the residents of the environs who gave testimony complained of daily, unremitting, tingling of the extremities. It was an opinion not shared by the other Alcolac expert, Kerby, who concluded that, the 3% incidence of such symptoms in the average population considered, the 78% statistic - "if indeed true" - is "different than the usual."

The two Alcolac medical experts, Emmett and Kerby, then entered separate diagnoses for each of the plaintiffs. These determinations rested on the examination reports, findings on physical examination, results of laboratory tests and panels, and on the personal, occupational and medical history of each compiled under the CCA regimen. The diagnoses of Emmett and Kerby rested also on the interview examinations and histories and on the selective laboratory test results administered among the plaintiffs under the K.U. protocol. These diagnoses did not include evaluations of the immune panels administered by Bioscience, Midwest/-Wheeler and K.U. Medical Center. That element of evaluation was left to immunologist Dr. Stechschulte, as our opinion already reports.

The function of the environmental medicine physician, Emmett explained, is less to treat by therapeutic care, than to establish a causal diagnosis for a bodily state. The end of such diagnosis is to determine "not only what somebody has, but why it

is caused." In that exercise, cause [or] etiology and diagnosis are ineluctable. The etiology may be an external event [such as a toxic exposure] or an occupational source, or a preexistent physical disease, or a life habit - among others. It is because there may be "hundreds of different causes" for a physical complaint or symptom that the history given by a patient is the most important single component of diagnosis. The medical cause is derived through the method of differential diagnosis which discards possible causes for the same symptom or condition for the cause which the physical findings, laboratory tests, history and best scientific data indicate with sufficient probability explain the symptom or condition.

A logical temporal relationship between the event and the physical effect bears significantly to validate causation. Thus, an irritant dermatitis which manifests the day after exposure - Dr. Emmett explained - tends to establish the exposure as the cause of the dermatitis. A cancer which appears the day after the chemical exposure, on the other hand, does not, since from what is known there was not enough time to develop cancer. It was just such an incidence of logical temporal relationship between the Alcolac odor and physical discomforts given as history by the several plaintiffs to the physicians that prompted Dr. Emmett to diagnose that the exposure to the Alcolac chemicals was the cause:

> "I think an example would be the ear, eye, nose and throat irritation, all the people, all--or a large number of people relating that to the chemicals, what they perceive to be the chemical odor. . . . That is the basis on which I made those diagnoses."

These medical opinions assumed for purposes of diagnosis that no alternative point source [such as the Missouri Pacific Railroad operation] was a cause of the symptoms related by the histories of the several plaintiffs.

The two expert physicians for Alcolac, Emmett and Kerby, then proceeded to enter their separate diagnoses as to each plaintiff. Those renditions juxtaposed the subelements of the Carnow diagnosis with their own and explained any disagreement.[52] The diagnoses entered by Emmett and Kerby were attributed either to: Alcolac, Another Cause. Pre-existing, Unknown. These opinions of diagnosis and etiology all issued on reasonable medical certainty. In the formulation of that medical opinion, the validity of the test procedures administered or conducted under the auspices of CCA was not usually in question. Nor was the integrity of the results. Emmett and Kerby did not equate an abnormal test result [one outside the reference range] with abnormal function. They came to determination of etiology and cause of a given bodily condition, rather, from the clinical significance of the test results considered together with occupational and medical history, and other such data.

Alcolac was entered as the cause of the manifestations of eye, nose, throat and skin irritations described by the plaintiffs, based upon - as Emmett and Kerby explained - the histories given them by the plaintiffs and the validity of the temporal relationship between the cause and effect. There was disagreement in some cases whether the diagnosis of a given manifestation - such as conjunctivitis, rhinitis or pharyngitis - was properly chronic [as entered by Carnow as to some of the plaintiffs] or simply recurrent.

As to virtually every other manifestation of bodily damage, organ dysfunction or disease, the etiology Kerby attributed was Another Cause, Pre-Existing, or Unknown.[53] Thus, the

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[52] See Appendix Q-1, Q-2, Q-3, typical diagnosis charts as composed by Dr. Emmett and explained in his testimony.

[53] The testimony of Dr. Kerby can be understood to attribute to Alcolac not only the complaints of eye, nose, throat and skin irritations given in the histories of the plaintiffs, but also [in the cases of Betty Elam, Gwendolyn Lawrence and John Phil-

abnormal audiometric test results from which Carnow diagnosed hearing loss from toxic chemical damage to the 8th cranial nerve for numerous of the plaintiffs, Emmett and Kerby attributed to presbycusis, or occupational noise, or - in one or two - some pre-existent conductive loss. The sources for these opinions of non-Alcolac etiology were from the medical records and personal histories of the plaintiffs. They were often cited, but not elaborated.[54] No instance of male infertility or low sperm count was attributed to Alcolac. Those dysfunctions found in Daniel Pryor were simply attributed to Unknown. There was no porphyrin-related disease diagnosed in any of the plaintiffs, whatever the etiology, although Emmett acknowledged that 27 of the 31 plaintiffs tested outside the normal reference ranges. Nor was there any liver dysfunction or disease, or any other of the several manifestations subsumed under the Carnow diagnosis of chronic systemic chemical intoxication attributed an etiology other than to Another Cause, Pre-Existing, Unknown. Dr. Kerby acknowledged, however, that the attribution of etiology Unknown could include toxic chemicals.

The prognosis entered by Dr. Kerby for each of the plaintiffs was good, very good, or excellent, except for Mary Landon. Hers was guarded.

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lips] the complaints and conditions of peripheral neuropathy.

[54]For instance, Dr. Kerby attributed the hearing loss the audiometric tests detected in Carl Berry as caused by his duty during the Korean War on an aircraft carrier and his later occupation as the operator of heavy equipment. That attribution of etiology assumed exposure to jet aircraft and lack of protective headgear in the naval occupation and noisy equipment in the civilian occupation. Carl Berry testified in rebuttal, however, that the aircraft were propeller driven, that he wore protective headgear, that he was tested monthly for consequences of his duty on his hearing, and that his hearing on final examination prior to discharge tested normal. His civilian occupation thereafter, he also testified, involved no unduly noisy equipment.

## VII

## Submissions, Verdicts, Judgments

The causes of action were ready for submission after eighty days of actual trial testimony. The litigation was originally brought as thirteen separate suits by thirteen family groups with a total of thirty-two suitors [55] These suits were consolidated on the motion of the defendant Alcolac and over the objection of the plaintiffs. They were tried to the jury as a consolidated litigation. To facilitate review and argument by counsel to the jury of the mass of testimony [witness as well as technical] the trial court announced the design to submit each of the thirteen suits separately. The submission of the first of the plaintiff groups would include the general instructions [meant to cover all thirteen submissions] and the specific instructions as to that first plaintiff group submission. The arguments to the jury would follow, the returns of verdict as to that group, and entry of judgment. Counsel would be allowed full argument to the first plaintiff group submission, and limited argument as to each of the others in sequence.

Alcolac objected to the submissions design on grounds that the intended method violated MAI and otherwise imposed undue burdens on posttrial motion and appeals procedures, and on the jury. Alcolac sought, and had the writ of this court to prohibit the trial judge from implementation of that design. In consequence, the thirty-one claims for personal injury and eleven claims for property damage were submitted and argued together.

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[55] As we noted, a directed verdict was entered against plaintiff Edward Gehlken at the close of the case for the plaintiffs. He had been incapacitated for reasons other than any misconduct by Alcolac, was not able to appear for the numerous examinations, and so failed to prove that he suffered any bodily injury by Alcolac. His nuisance claim for damage to the property he owned with Malva Gehlken remained intact.

The jury awarded to each plaintiff on the claims for personal injury, $200,000 as compensatory damages and $1,387,096.70 as punitive damages. There were separate awards returned for the property damage. Alcolac moved for judgment notwithstanding the verdicts or, alternatively, for new trial. The trial court denied judgment n.o.v., but granted a new trial as to damages only as to each of the thirty-one personal injury verdicts.

On the issue of compensatory damages the formal order recited as ground for new trial that the return of an identical $200,000 verdict for each plaintiff was

> impermissible as a matter of law . . . and are inconsistent with and irreconcilable to each other, when viewed in context with all of the evidence favorable to the thirty-one plaintiffs

On the issue of punitive damages the formal order recited as ground for new trial, inter alia, that

> [t]he aggregate amount of the punitive damages awarded exceeds by almost three times the net worth of the defendant, and thus is impermissible as a matter of law because such verdicts have the effect of confiscating property without due process of law.

The memorandum opinion appended to the order made emphatic that the grant of new trial was not an exercise of the discretionary power of the court, but that these rulings rested "on legal grounds alone":

> Such a conclusion is not predicated on the sufficiency of the evidence or the weight thereof, but solely on the illogical nature of the verdicts as compared to the evidence and each other.

Alcolac appeals from the grant of a new trial on the issues of compensatory and punitive damages for personal injury and from the denial of judgment notwithstanding verdicts on the claims

of all of the plaintiffs. The plaintiffs appeal from the grant of a new trial on the issues of compensatory and punitive damages and seek reinstatement of the verdicts as rendered by the jury.

## PART TWO

### The Alcolac Appeal on the Negligence Causes of Actions

Alcolac seeks alternative relief on appeal: entry of judgment notwithstanding the verdicts for failure of plaintiffs to prove submissible negligence and nuisance causes of action, or remand of the consolidated cases for new trial on all issues, rather than merely for damages on the negligence causes of action as ordered by the trial court.

The plaintiffs framed recovery on two theories, negligence and nuisance. One theory was that the negligence of Alcolac caused injury to the persons of the plaintiffs. The other theory was that the unreasonable use of its property by Alcolac caused injury to the property of the plaintiffs. Alcolac insisted at the trial, and insists now on appeal, that there was no competent proof that either any injury to the persons of the plaintiffs or to their property was caused by Alcolac. Actionable negligence requires competent proof that the injury for which the damages are sought was caused by the defendant. Virginia D. v. Madesco Inv. Corp., 648 S.W.2d 881, 886[4,5] (Mo. banc 1983). Actionable private nuisance imposes a like proof of causation. Fuchs v. Curran Carbonizing and Engineering Co., 279 S.W.2d 211, 219[16,17] (Mo. App. 1955).

The dominant theme of the trial was the claim that Alcolac negligence caused biological disease and dysfunction to the plaintiffs. The proof of causation in the negligence cause of action, therefore, although somewhat coincident with the proof of that element in the private nuisance [unreasonable interference

with the enjoyment of property] claim, requires a more extensive and intricate proof. It subsumes the other. For that reason, we address only the contention that judgment notwithstanding the verdicts must be entered because there was no competent proof that Alcolac negligence caused the biological conditions the plaintiffs describe in the evidence. Our response to this contention as to the negligence cause of action determines the validity of the submission of the nuisance cause as well.

Actionable negligence is proven by evidence of a legal duty on the defendant to protect the plaintiff from injury, failure of defendant to perform the duty, and injury to the plaintiff caused by such neglect. Hoover's Dairy, Inc. v. Mid-America Dairymen, 700 S.W.2d 426, 431[1,2] (Mo. banc 1985). The plaintiffs pleaded a duty owed them by Alcolac not to store, emit, dump or otherwise dispose of toxic chemicals, wastes, pollutants or contaminants which they reasonably knew or should have known would cause harm to the plaintiffs, and rather, to contain these toxic contaminants and pollutants in a reasonable manner, or warn adjacent dwellers of these activities so that they could take protection against them. The plaintiffs pleaded breaches by Alcolac of the duty to protect them from injury, and pleaded also the incurrence of personal injury to them caused by the breach of the duty. Alcolac does not contend that it was under no duty to protect the plaintiffs from harm, nor that the evidence does not prove prima facie breaches of that duty. Alcolac confines the assertion for judgment notwithstanding the verdicts to the contention that negligence was not submissible because there was no evidence that any breach of duty caused bodily injury to any plaintiff. That is to say - that cause in fact between any Alcolac conduct and damage to any plaintiff was not proven.

I
A
Judgment Notwithstanding the Verdicts

1.
The Issue of Causation in Fact
The Principles

An essential element of the proof of a cause of action for negligence, or any other tort, is that there be some reasonable connection between an act or omission of the defendant and the damage the plaintiff has suffered. This connection is the "causation in fact" of the damage. Prosser and Keeton, The Law of Torts § 41 [Fifth ed. 1984]. The proof of the causation in fact element in a toxic tort case is distinctively different from that in the traditional action for personal injury. In the conventional paradigm, A is struck and injured by the vehicle of B - the injury follows upon impact. In such a case, the cause is a single definable event and the effect is direct, immediate and observable. The connection in fact between the conduct of the defendant and the injury to the plaintiff is readily deducible, and thus not usually a matter of dispute. A typical toxic tort case, on the other hand, involves a claim of personal injury related to harm from exposure to a toxic substance - usually a chemical. The exposure is typically chronic and repeated, the injury is not from trauma or acute toxic response but from genetic or biochemical disruption, and manifests only after a long period of latency. Ayers v. Jackson Tp., 525 A.2d 287, 301 (N.J. 1987); Gold, Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence, 96 Yale L.J. 376 (1986). The cancer and other diseases manifested from exposure to toxic agents, moreover, are found to occur at background levels in other segments of the population, even without apparent cause. Black & Lilienfeld, Epidemiologic Proof in Toxic Tort Litigation, 52 Fordham L. Rev. 732, 744 (1984). The latency factor as well as the background

incidence of the disease the claimant attributes to the toxic agent engender a causal indeterminancy which hampers the ability to isolate the substance which induced the injury. Allen v. United States, 588 F. Supp. 247, 404 et seq. [60] (D. Utah 1984), rev'd on other grounds, 816 F.2d 1417 (10th Cir. 1987); cert. denied, ___ U.S. ___, 108 S.Ct. 694 (1988); Harris, Toxic Tort Litigation and the Causation Element: Is There Any Hope of Reconciliation, 40 Sw. L.J. 909, 912 (1986). Thus, the logical model of a single definable cause and a direct, immediate and observable [and hence, determinate] effect that suffices to prove cause in fact in the traditional tort cause of action does not suit the toxic tort explanandum.

A tort claimant must nevertheless prove a factual connection between the conduct of the defendant and the injury or suffer adverse judgment. Our law regards conduct as cause in fact [otherwise, legal cause] of harm if "the injury would not have occurred in the absence of the negligent act" [Dintelman v. McHalffey, 435 S.W.2d 633, 636[1,2] (Mo. 1968)], or, alternatively, if the conduct "is a substantial factor in bringing about the harm" [Ricketts v. Kansas City Stockyards Company of Maine, 484 S.W.2d 216, 221[4-6] (Mo. banc 1972)]. The "but for" rule [which Dintelman and a strand of our decisions employ as the test of causation in fact] is by function a rule of exclusion and as such "serves to explain the greater number of cases." Prosser and Keeton, The Law of Torts § 41, at 266 [Fifth ed. 1984]. There is, however, one type of situation in which it fails: "If two causes concur to bring about an event, and either one of them, operating alone, would have been sufficient to cause the identical result, some other test is needed." Id. The substantial factor test is the response. In the operation of this principle, "'substantial' [denotes] the fact that the defendant's conduct has such an effect in producing the harm as to lead reasonable men to regard it as a

cause." Giles v. Moundridge Milling Co., 351 Mo. 568, 173 S.W.2d 745, 750[6-8] (1943); Restatement (Second) of Torts § 431, comment a (1965). Our law adopts the scope Restatement (Second) of Torts § 432(2) accords the substantial factor principle:

> If two forces are actively operating, one because of the actor's negligence, the other not because of any misconduct on his part, and each of itself is sufficient to bring about harm to another, the actor's negligence may be found to be a substantial factor in bringing it about.

Giles v. Moundridge Milling Co., 173 S.W.2d at 750; Chism v. White Oak Feed Company, Inc., 612 S.W.2d 873, 881 (Mo. App. 1981). The substantial factor rule is acknowledgment of the reality that, in the usual course, "[an] event without millions of causes is simply inconceivable; and the mere fact of causation, as distinguished from the nature and degree of the causal connection, can provide no clue of any kind to singling out those which are to be held legally responsible." Prosser and Keeton, The Law of Torts at 266.

Thus, although our law requires proof of cause to recover in tort, it does not require proof of a single cause. The substantial factor standard - which ascribes liability to a cause which has played an important part in the production of the harm, even though the harm may have occurred absent that cause - is particularly suited to injury from chronic exposure to toxic chemicals where the sequent manifestation of biological disease may be the result of a confluence of causes. Allen v. United States, 588 F. Supp. at 418; Basco v. Sterling Drug Co., 416 F.2d 417 (2d Cir. 1969); Gold, 96 Yale L.J. 391; Harris, 40 Sw. L.J. 911; M. Dore, Law of Toxic Torts, Environmental Law Series, § 24.02 (1982).

A toxic tort plaintiff, as any other, bears the burden to prove the facts essential to a prima facie case. MAI III 3.00 [1987]. The plaintiff also bears the risk of nonpersuasion and

must show by a preponderance, or greater weight, of the evidence that the injury was the result of the negligence of the defendant. Ledkins v. Missouri-Kansas-Texas Railroad Company, 316 S.W.2d 564, 568[5] (Mo. 1958); Rexford v. Philippi, 337 Mo. 389, 84 S.W.2d 628, 632[5] (1935). The greater weight of the evidence does not mean evidence which engenders certainty in the trier of fact, but which - "when the last word has been spoken in the trial of a case" - prompts a greater probability of confidence in the evidence of one party over the other. Brown v. Sloan's Moving and Storage Company, 274 S.W.2d 310, 313 (Mo. 1954); Miller v. Watkins, 355 S.W.2d 1, 2[1-3] (Mo. 1962); Kenney v. Henson, 107 S.W.2d 947, 952[1] (Mo. App. 1937). "[A] standard of proof represents an attempt to instruct the factfinder concerning the degree of confidence our society thinks he should have in the correctness of factual conclusions for a particular type of adjudication." In re Winship, 397 U.S. 358, 370 (1969) [Harlan, J. concurring]. The probability which induces the verdict of the jury in favor of one party over the other, therefore, is not a mathematical value, but a state of confident belief.

Alcolac does not dispute that it owed a duty to the population in the environs to protect them against the hazards from the careless release into the environment of toxic pollutants and other residues of their production process. That was the very purpose of the systems of filtration, scrubbers, incinerators and other environmental control devices the plant design intended. Nor does Alcolac genuinely dispute that the evidence suffices to protect the plaintiff-members of the population in the environs from that risk of harm. The replete evidence - of a liquid incinerator which only partially consumed the toxic contaminants so that countless pyrolysis compounds of equal or more toxic effect were expelled into the atmosphere, of the recurrent diversion of the toxic stream around the filtration system directly into the

bioponds so that the contaminants evaporated into the atmosphere, of the habitual overload of the bioponds with the organic wastes from the monomer process so that the contaminants [toluene, methyl chloride and epichlorohydrin, among others] were borne into the ambient air so as to expose the persons in the environs to these toxicants throughout the eight-year course of Alcolac operations - suffices prima facie to prove breach of duty.

2.

The Issue of Causation in Fact

The Argument

Alcolac insists, rather, that the actions for negligence were not submissible because causation in fact was not proven as a matter of law, in particular, that there was no competent proof that:

1. any plaintiff ever inhaled, ingested or absorbed any injury-causing substance emitted from the Alcolac premises

2. Alcolac ever emitted from its premises an injury-causing substance that injured any plaintiff

3. any injury-causing substance was ever identified as emitted by Alcolac so as to injure any plaintiff

4. the times or duration of time when any plaintiff inhaled, ingested or absorbed any injury-causing substance were ever identified

5. the amount of any injury-causing substance inhaled, ingested or absorbed by any plaintiff was ever identified

6. any act or omission of Alcolac ever caused injury to any plaintiff

7. any act or omission of Alcolac was ever linked or connected to any injury of any plaintiff.

Alcolac argues urgently that since modern life exposes everyone to countless chemicals and other toxic residues from diverse sources, a plaintiff who claims injury from such an exposure must identify the particular substance which caused the injury, as well as the source. Alcolac acknowledges that circumstantial proof of cause suffices, but argues that the evidence presented by the plaintiffs, however massive, was not sufficiently probative to interstice that element essential to recovery. That argument says that no lay, scientific or medical evidence identified an "injury-producing substance or emission," or the person responsible for such substance or emission, or that the etiology of any bodily condition claimed by any plaintiff was attributable to Alcolac.

Alcolac comes to the view of the uselessness of the plaintiffs' evidence on the premise that proof of causation "requires not only proximity in time, but also a showing that plaintiff[s] would not have been injured but for defendant's negligence." That, as we note, is the standard of causation our law expects in the greater number of cases where the cause is a single identifiable event and the effect is direct, immediate and observable. In such cases, the law insists on particularistic evidence of causation. Dintelman v. McHalffey, 435 S.W.2d at 636[1,2].[56] In this case, as the evidence shows, the event was not single, but an exposure recurrent over as long as eight years, and some of the effects, the experts say [increased risk of cancer, continued deterioration of the body organs and of the immune system], remain latent. Also, genetic and other causes may impinge so that particularistic evidence of a cause in close temporal relationship with the effect is beyond the plaintiffs to muster. In a word, the chronic nature of the conduct of the toxic

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[56] See decisions listed in the Missouri Digest 2d Negligence § 56(1.2) Test for causal relation.

polluter - Alcolac - and the indeterminacy that the toxic pollution was the exclusive source of the disease prevent such a plaintiff from the proof that but for the Alcolac conduct the bodily injury would not have eventuated. Rosenberg, The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System, 97 Harv. L.Rev. 851, 855 (1984).

The but for formula, no less than substantial factor, is as much an expression of legal policy as of factual quantum. Prosser and Keeton, The Law of Torts § 41, at 256 (Fifth ed. 1984); Harris, 40 Sw L.J. at 952. The traditional and foremost policy of the tort law is to deter harmful conduct and to ensure that innocent victims of that conduct will have redress. Cognate principles of equity and economic efficiency also inform that policy: that the costs of the pervasive injury which result from mass exposure to toxic chemicals shall be borne by those who can control the danger and make equitable distribution of the losses, rather than by those who are powerless to protect themselves. Elmore v. Owens-Illinois, Inc., 673 S.W.2d 434, 438 (Mo. banc 1984); Morrow v. Caloric Appliance Corporation, 372 S.W.2d 41, 54 (Mo. banc 1963). The formulas of causation in fact are means to those ends and accommodate the stringencies of evidence to the policy they subserve. In that sense, causation in fact interdepends with duty, breach of duty and proximate cause. See Zafft v. Eli Lilly and Co., 676 S.W.2d 241 (Mo. banc 1984); Keener v. Dayton Electric Manufacturing Company, 445 S.W.2d 362, 364 (Mo. banc 1969); Howarth, Causation and the law, 96 Yale L.J. 1389, 1419 (1987); G. Calabresi, Concerning Cause and the Law of Torts: An Essay for Harry Kalven, Jr., U. Chi. L. Rev. 43, 69 et seq. (Fall 1975).

Alcolac argues cause in fact - not in the context of operational practices which virtually precluded evidence to an injured suitor - but in terms of lack of essential proof that very evidence would have enabled. It was evidence of a state of the

art system of toxic waste control and disposal mismanaged by untrained personnel and by operating procedures left undefined. It was evidence of recurrent toxic spills vented into the atmosphere, but unrecorded so that the quantity, identity and dates of those exposures cannot be known. It was evidence of toxic waste from the monomer process diverted from the decontamination procedure and expelled, still toxic, into the bioponds for evaporation into the air - without record or other note of these recurrent events. It was evidence of a liquid incinerator, designed to consume particulate toxic wastes, but which so malfunctioned as to be declared a "hundred percent failure" by the Alcolac management - but continued in use for more than two years so that countless, unidentified, unmeasured and unrecorded emissions of toxic compounds were released into the atmosphere. It was evidence of an agreement by Alcolac with DNR [May 30, 1980], but never honored, to install monitors on the monomer stacks to identify and measure the emissions for each product - so that the record intended by the regulatory agency never took form. Thus, the but for proof Alcolac insists upon the toxic tort plaintiffs [even were such a proof otherwise open to them] was made impossible by the very conduct of the defendant. These neglectful, even heedless, business and production practices, moreover, burden the plaintiffs in the proof that the Alcolac conduct was a substantial factor as a cause of their harm - that is to say: that the Alcolac conduct "ha[d] such an effect in producing the harm as to lead reasonable men to regard it as a cause." Giles v. Moundridge Milling Co., 173 S.W.2d at 750[6-8]; Restatement (Second) of Torts §§ 431, 432 (1965).

Alcolac, in any event, confounds the effect of its own evidence by the contention that no diagnosed injury bore that "proximity in time" to the emissions essential to the proof of a but for causation. It was the testimony of Alcolac expert Emmett

that in environmental medicine cause and diagnosis are inextricable. A logical temporal relationship between the event and the biological effect, he explained, bears significantly to validate cause [or - in medical terms - etiology]. An irritant dermatitis which manifests the day after exposure [as was the case with several plaintiffs] is such a logical temporal relationship as to establish the exposure as the cause of the dermatitis. On the other hand a cancer - which invariably requires a long period of latency - which appears soon after exposure to the toxicants does not display a logical temporal relationship between cause and biological effect. It was the evidence that the plaintiffs were subjected to chronic exposure of the Alcolac emissions - some of them for as long as eight years.[57] The experts at the trial agreed with the other authorities on toxicology that: "Chronic exposure

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[57]The exposures to the Alcolac emissions to which the plaintiffs attribute their bodily diseases spanned the period from commencement of plant operations in May of 1978 to the commencement of trial in August of 1985 - an interval of seven years. Only eight of the plaintiffs were subjected to exposure for the entire period. The duration of exposure varied as to the other twenty-four plaintiffs. Carl and Jacqueline Berry took up residence in the environs of Alcolac in 1979, so the maximum period of their exposure was six years. Virgil and Dorothy Bradley moved into Sedalia proper in 1984 when the odors and emissions did not abate - [a six-year exposure]. Clarence Elam was employed away from home during the week from 1978 to 1979, and returned home only on weekends - [an enhanced six-year exposure]. Linda Elam Sanders married in 1980 and removed to Texas - [a one-year, nine-month exposure]. John and Gwendolyn Lawrence were prompted by the odors and emissions to leave the premises in November of 1983 for residence on the far side of Sedalia, and lived intermittently with a nearby daughter - [a diminished five-year exposure]. Dainie and Mary Landon left the residence in May of 1984 to escape the emissions and moved into a mobile home - [a six-year exposure]. Glenn and Bernice Miller removed to Springfield in August of 1984 to escape the odors - [an enhanced six-year exposure]. Daniel and Joyce Pryor, with her daughter Amber, left the residence in February of 1984 after the dissolution of the marriage - [an enhanced six-year exposure]. Tammy Sommers left the family residence in January of 1983 - [an enhanced four-year exposure]. The Turley family - James, Kay, Lance, Lisa and Lyle - were prompted by the Alcolac odors to remove to Texas in May of 1981 - [a three-year exposure].

to a toxic agent may produce immediate or acute effects with each administration in addition to the long-term, low-level, or chronic effects of the agent." Casarett and Douell's Toxicology 14 (2d ed. 1980).

It is not disputed in the evidence that, based upon the symptoms and history of complaints the plaintiffs reported to defense physicians Emmett and Kerby and the sequence between the incidents of recurrent exposure to the Alcolac odors and emissions and the manifestations of eye and upper respiratory irritations, these examiners assigned Alcolac as the cause. That is to say: the recurrent sequences of exposure to odors and the acute effects were in that "proximity in time" to constitute that but for nexus Alcolac insists on, between any tortious conduct and injury.

Alcolac argues nevertheless that there was no competent evidence of causation - as to either acute or chronic effects - attributable to its operations. Alcolac insists that no competent evidence was adduced that Alcolac ever emitted any harmful substance any plaintiff ever inhaled or otherwise took into the bodily system, or ever caused any injury to any plaintiff. We consider that contention - as it impinges on the recurrent complaints of eye irritation, nose, throat and other upper respiratory ailments which manifested in that "logical temporal relationship" between exposure and effect expected as a validation of causation by Dr. Emmett - an issue conceded by the defense evidence. It was qualified by the proviso only that no source of the odors or emissions alternative to Alcolac be shown as the cause. That proviso, as our discussion determines, was not established. The method of diagnosis employed by both Emmett and Kerby, it is to be understood, was the usual procedure they employed to reach medical cause - and hence diagnosis. It rested on the numerous histories to be sure, but also on information of the Alcolac production process, the toxicological profiles of the chemicals

used, on the profuse medical records gathered by the plaintiffs, and the reports under the K.U. protocol. In short, as Dr. Emmett acknowledged: "I made those diagnoses on the information that I reviewed" - as well as the additional information given at the trial.

A plaintiff who claims harm from exposure to a toxic substance bears the risk of nonpersuasion that the conduct of the defendant was a substantial cause of the injury. Basko v. Sterling Drug Co., 416 F.2d 417, 429[12] (2d Cir. 1969); Restatement (Second) of Torts § 432(2) (1965); Giles v. Moundridge Milling Co., 173 S.W.2d at 750. In a toxic tort case, the cause in fact is a manifold proof: [1] an exposure to an identified harmful substance significant enough to activate disease [2] a demonstrable relationship between the substance and biologic disease [3] diagnosis of such disease in the plaintiff [4] expert opinion that the disease found in plaintiff is consistent with exposure to the harmful substance [5] defendant was responsible for the etiologic agent of the disease diagnosed in plaintiff. 2 Toxic Law Reporter (BNA) [hereinafter TXLR] 1040 (1988); M. Dore, Law of Toxic Torts § 24.03 (1987). The element of significant exposure to the harmful agent once established, the question becomes whether the pollutant [or other substance] can be demonstrated to cause the type of harm the plaintiff reports, and whether the harm did in fact result from the exposure. The diagnosis of disease induced by environmental factors [the biologic causation elements of causation in fact] is essentially a scientific undertaking, and so the burden of proof of causation the law imposes on a toxic tort plaintiff must be accommodated to the quality of evidence the scientific community deems sufficient for that causal link - if such a plaintiff may have recovery at all. Gold, 96 Yale L.J. at 384; Harris, 40 Sw L.J. at 945.

Alcolac insists however that there was no competent proof that Alcolac ever emitted any injury-producing substance, or that any plaintiff ever took any such substance into the bodily system, or that any plaintiff ever identified any such substance, or the times and duration of exposure, or the dosage taken in - or that any Alcolac conduct ever caused injury to any plaintiff. That is to say: none of the elements of causation in fact were proven by competent evidence, and the cause of action was not submissible.

Indeed, no witness - expert or lay - could identify the particular chemical at a particular exposure, the particular concentration of the chemical, the particular dosage of the chemical taken in bodily, or the particular duration of the exposures.[58]

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[58] In fact, as early as 1978, a number of the plaintiffs commenced and kept calendars of the incidence of odors, haze, foam and other emissions from Alcolac - as well as of the episodes of burnings on the Alcolac premises. The entries were not uniform. Some describe the odors. Some note the duration of the odors and emissions. All record the dates of those events. The entries are intermittent - and presumably coincided with the dispersion of the pollution by the variable winds in that locale. The diary of Dainie and Mary Landon commenced in 1978 and records the incidence of odors as early as June 13th of that year. The Landons also kept a diary of the physical symptoms and manifestations the diarists attributed to the exposures, as well as the medical treatment received for those conditions. The plaintiff chroniclers include Ethel Berry, Carl and Jacqueline Berry, the Bradleys, the Elams, the Landons, the Lawrences, the Phillips, the Sommers and the Withers.

The incidence of these odors, emissions and residues from Alcolac recorded in the calendars was on the average of fifteen days per month. The Berry, Bradley, Elam and Landon residences are sited on three sides of Alcolac - where all of the plaintiffs were located, except for the Pryor family. Virtually all of the fifty nonplaintiff residents in the environs, who also testified concerning the Alcolac emissions, are sited on similar - but more extended - radii from the plant. [See Appendix B] Those plaintiffs who did not keep calendars, as well as the fifty nonplaintiff residents in the environs, testified to the same odors, the same emissions, frequency, and to the same physical effects. Some of these nonplaintiffs lived more than three miles from the Alcolac plant.

The direct proof of the identity of the toxic agents emitted by Alcolac over more than seven years, their measurements and frequency - which the defendant contends was essential to evaluate the role of those chemicals in the assessment of the claims of risk and injury - was evidence made unavailable by the insistent neglect of Alcolac to install monitors on the stacks and by the heedless, continued use of the liquid incinerator for more than two years after its malfunction. The lack of monitors at each single point source, a condition which continued up to the time of trial more than five years after the agreement with DNR to install them, deprived the plaintiffs of the identity of each chemical compound emitted, the concentration, and the frequency as well. Moreover, the imperfect incineration of the toxic waste from the malfunction not only distorted the identification of the compounds intended for destruction by that method, but also resulted in a profusion of pyrolysis products of unknown kind, quantity and toxicity.

The identity of the toxic substances to which the harm is attributed, however, may be shown by circumstantial evidence. Roehling v. National Gypsum Company Gold Bond Building Products, 786 F.2d 1225, 1228[2,3] (4th Cir. 1986); Drayton v. Jiffee Chem. Corp., 395 F. Supp. 1081, 1087 (N.D. Ohio 1975), modified and aff'd, 591 F.2d 352 (6th Cir. 1978). The toxic compounds to which the plaintiffs attributed their diseases were epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, glycidyl ether, cyclohexane, dimethyl sulfate, hydrogen sulfide, methyl chloride and allyl methacrylate. The average quantity of each of the first five of these chemicals used by Alcolac every year for seven years was also known and calculated. The quantity and kind of chemicals used in the monomer process varied from batch to batch, but these few were designated by the plaintiffs because of the many compounds used by Alcolac, the toxic profiles of these ten were well

delineated. The evidence does not dispute that the chemicals in these quantities were used in the Alcolac production processes and that their wastes were intended for disposal in the environmental control systems installed in the plant. Nor was the evidence contradicted that the tests of the bioponds measured the footprints of epichlorohydrin in the form of breakdown products, of toluene and of methyl chloride - three of the compounds identified as factors of disease. The testimony of former Alcolac employees, as confirmed by the official spill log briefly maintained at the plant, also identify quantities of these chemicals spilled and then vented into the atmosphere, or simply routed directly into the bioponds without prior decontamination. Then also DNR investigators traced odors to spills of allyl alcohol on the Alcolac premises and emissions from other of these identified chemicals from that source. These circumstances in evidence sufficed to prove the identity of the toxic substances to which the plaintiffs attributed their injuries.

Alcolac complains that measurements of the toxic agents lacked, and hence causation in fact of any injury was not proven. The most apt manner to measure concentrations of chemicals in the air is by the monitor. The personal monitor directly measures the contaminants in the air which the individual breathes. The efficacy of the personal monitor depends upon data from a study group. The monitor on a plume or stack [as intended by the May 1980 agreement between DNR and Alcolac] directly measures the emissions for each single point source. The efficacy of a stack or biopond monitor depends upon its installation. Office of Science and Technology Policy, Chemical Carcinogens: A Review of the Science and Its Associated Principles, 50 Fed. Reg. 10425 (Feb. 1985). A

person subjected to chronic, long-term chemical exposures is not expected to foster a study group in order to gather evidence to prove liability for a disease not even anticipated.[59] An industrial polluter who refuses to make possible the measurement of the pollution - despite official obligation to that end - cannot complain that the measurement lacks. In any event, measurements of concentrations are useless as factors of exposure assessment without information of the identity of the chemicals measured, and Alcolac foreclosed that data. Allen v. United States, 588 F. Supp. at 425.

This lack of detail of exposure quantitation notwithstanding, the evidence agrees that the plaintiffs were exposed on sufficient occasions, for sufficient durations, in sufficient concentrations to toxic Alcolac chemicals to cause them recurrent [and even chronic] irritations and ailments to their eyes, skin and upper respiratory systems. There was other evidence, moreover, relevant to the proof of the frequency, duration and concentrations of exposure, of the effects of the emissions on the fauna and flora in the environs. The testimony of the residents in the Alcolac environs, plaintiffs and nonplaintiffs alike, of chemical residues on the gardens and trees, followed by decay and destruction - of visible physical effects of these emissions on the domestic animals and pets, of the fish kills in the waters mixed with the Alcolac effluents, all bore relevantly as circumstantial evidence of the frequency and recurrence of the episodes of emissions, as well as of concentrations sufficient to those effects.

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[59] Actually, not too long after the emissions became bothersome, DNR installed a "personnel sampler" furnished by the EPA in an appurtenant building on the Landon premises. A logbook was left with instructions that the Landons go to that structure and turn on the pump whenever they smelled odors and record the time as well as the time the odors ceased. The pump was then to be turned off and the time also recorded. EPA required the return of the sampler after a month, and the pump had never been turned on.

In this state of the evidence, testimony of the "total adverse effects produced by the toxicant when administered continuously over a long period of time" bears as circumstantial proof of causation. See J.H. Duffus, Environmental Toxicology 5 (1980); Hall and Silbergeld, Reappraising Epidemiology: A Response to Mr. Dore, 7 Harv. Envtl. L. Rev. 441, 445 (1983).

Alcolac argues also that causation in fact was not proven because no competent evidence shows that "any plaintiff ever inhaled, ingested or absorbed" any injurious substance from the Alcolac source. We understand that argument to mean that, whatever the duration, concentration or frequency of any emission of toxicants by Alcolac, there was no evidence that any impinged on any of the persons of any of the plaintiffs so as to cause any of them injury. The evidence easily refutes that contention.

The odors generated by the Alcolac operation of its bioponds, liquid incinerator and production practices and their effects on nearby residents became matters of concern to the management virtually from the onset of activity. Operation commenced in May of 1978 and in June of 1978 Alcolac received complaints from nearby residents of vapors and haze over their properties. Among the complainants were the plaintiffs Phillips. WAPORA had been engaged to investigate this and other complaints. The vapors and haze were traced to the malfunction of the liquid incinerator. WAPORA engineer Boies came to the Phillips residence and apologized for those emissions. The citizenry became so disquieted by the persistent exposures to the vapors and haze generated by the liquid incinerator that WAPORA head Bregman was prompted to meet with them in public and reassure them that "these things were going to be solved." The incidence of continued pollution prompted Bregman once again to meet with the citizenry, this time with an environmental committee, to reassure them that the air pollution problems would be resolved. The citizens,

plaintiffs Landon, Phillips and others among them, continued to experience odors with consequent bodily discomforts and complaints, and continued to report them to Alcolac. At first, the management responded: Plant superintendent Fischer [a party defendant to the suit], his successor Sutton, his successor Aid, and even Alcolac president Anderson came to the home of plaintiffs Phillips to show concern. Those plaintiffs related the irritations to eyes and other bodily discomforts from the odors, vapors and foam. "They promised to do something about it." The Phillips couple [later plaintiffs] complained personally to Anderson on four occasions, and as noted on their calendar, Anderson came to their home in response on April 2, 1981. They described the odors and the consequent physical effects to them. Anderson told them Alcolac "w[as] going to do something about it." Alcolac plant superintendent Fischer acknowledged complaints from the nearby residents of odors from the Alcolac premises, and confirmed them. He attributed them to low general oxygen levels in the ponds, as well as "too much organic loading" of the ponds.

The recurrent complaints from the Landons, Phillips and other citizens of odors and other emissions from Alcolac ultimately came to the attention of DNR. A regimen of inspections and surveillance was established as already fully recounted in the narrative of the evidence. Numerous citations issued to Alcolac for excessive off-premises emissions and numerous violations were determined. A number of the incidents were acknowledged by Alcolac but attributed to shifting carbon beds or other malfunction pleas to avoid sanction. The complaints became so numerous and recurrent that plant superintendent Sutton refused to acknowledge them further. The abatement agreement of May, 1980 was the result.

There is simply no color of validity to the contention that no incident of Alcolac emission of a substance that could impinge harmfully on any plaintiff was proven. Even after the abatement agreement, Sedalia police officer Rice - to whom complaints were then referred for more expedited attention - received twenty-three separate complaints of odors, all of which were investigated and led to Alcolac. Alcolac itself notified Nikkila of DNR to report spills in order to explain complaints of odors. There was testimony of repeated chemical spills during the monomer production process which were either vented into the atmosphere or flushed down the drain for discharge, still toxic, into the biopond. There they mixed with the soap effluents from the sulfonation plant. For the year 1981 - the only period for which a separate log was kept - 111 spills were recorded. There was testimony, however, that the vast number of spills went unrecorded. There was testimony of regular diversions of raw chemical waste from the decontamination process directly into the bioponds because of overloaded sumps. The chemical spills as well as the diverted waste included those compounds already identified in the monomer production process: allyl alcohol, toluene, glycidyl ether, epichlorohydrin, and the others. The toxic qualities and the effect on the human system of those compounds were as ascribed by the toxicology experts Legator, Schwartz and Carnow.

There was other evidence by plaintiffs and nonplaintiffs alike - stated, iterated and reiterated - of periodic "foam storms" when suds formations from the bioponds were seen transported through the air beyond the bounds of the Alcolac premises onto grounds in the vicinity. The foam was an admixture of the sulfonation and monomer effluents and so - according to industrial hygienist Boelter - transported the monomer contaminants to wherever they alighted. There was evidence that this process of untreated discharge created a "soup" of toxic chemicals in the

bioponds which evaporated into the atmosphere. Toluene, methyl chloride and epichlorohydrin were identified by tests as among those toxicants contained in the bioponds - and hence to which the plaintiffs were exposed. Blowers, the only environmental engineer ever employed by Alcolac, acknowledged that foam accumulated on occasion to a height of 15 to 20 feet, and when the wind arose, blew all about and onto the property of the Alcolac neighbors. The testimony of chemical operator Faulconer was to the same effect. Faulconer also described clouds of acid mist emitted from the sulfonation plant stack when the acid scrubbers malfunctioned - a frequent phenomenon - which irritated the skin on touch. There was other evidence of explosions which blew chemicals into the atmosphere and, at least on one occasion, material on nearby property. These were among the numerous incidents of emissions beyond the Alcolac premises the evidence describes.

The calendars of the plaintiffs Berry, Bradley, Elam, Phillips and Lawrence record repeated sightings of foam from Alcolac transported onto their premises. The accumulations were described as "eleven feet high," "eight feet high," "as high as a refrigerator." The odors were "terrible" and the bodily effects upon contact were immediate. Mary Landon touched the foam and her skin blistered. Photographs of both the off-site accumulations of the foam were in evidence as well as of the physical consequences of contact with the substance. The odor, haze, smoke and foam phenomena were also described by nonplaintiff residents in the environs. The odors of the sulphur type, of rotten eggs, of septic or paint thinner they described, moreover, were the same off the Alcolac premises as on them where numerous residents had been on work or other occasions. Even momentary exposure to them, many related, resulted in irritation to the skin, mucous membrane, to the eyes, and even into the stomach. It was the testimony of Alcolac environmental medicine expert Emmett that the irritations

to the skin, nose, eyes and throat experienced recurrently by the plaintiffs mean that the odors and fumes "have contacted the surface tissues." Alcolac expert toxicologist Schwartz acknowledged that odors, fumes and vapors are "at least markers of a chemical agent" and, to the extent that the person has a symptom directly after exposure to the chemical agent, the "temporality" between the agent as cause and the symptom as effect is established. That is to say at very least, that a person who smells a chemical and thereupon feels its effect, is exposed to that chemical and impinged by it.

Nor, as Alcolac suggests, does Zafft v. Eli Lilly & Co., 676 S.W.2d 241 (Mo. banc 1984), require us to impose on those chronically exposed to toxic pollutants, unmeasurable because of the misconduct of the polluter, proof of the particular measurements of the chemical agents which caused their bodily injury as precondition to assessment of the risk of that exposure. That would only encourage the disregard of safe environmental practices and ensure escape from liability by simply flaunting official standards.

In any event Zafft treats the question of indeterminate defendant, not indeterminate measurement. Zafft, a products liability case, holds that a plaintiff may not maintain a cause of action in tort against a defendant the pleading cannot identify as an actor responsible for the substance which caused harm to the plaintiff. In Zafft, plaintiffs claimed injury from in utero exposure to DES, but were unable to identify the specific manufacturer from which they purchased the substance, so they joined as defendants all known manufacturers and distributors of the chemical substance from which DES derived. The plaintiffs argued a number of theories to sustain a recovery against the summary judgment entered on the pleading - among them, alternative liability, concert of action theory, market share liability, and its

variant. Zafft rejected each of them in turn. Alternative liability was not applicable, because [676 S.W.2d at 244]: "[u]nlike the typical situation warranting application of alternative liability, all possible tortfeasors are not before the court and the actual wrongdoers may escape liability." The concert of action theory was rejected by Zafft as a basis for liability because the required element of agreement among the alleged wrongdoers lacked. The market share liability notion was rejected because that theory [id. at 246] "continues the risk that the actual wrongdoer is not among the named defendants."

Here, as our fuller discussion concludes, Alcolac was the only reasonably possible point source of the pollution to which the plaintiffs were exposed - hence Alcolac was the only reasonably possible tortfeasor of the injury the plaintiffs claim, and Alcolac is before the court. Zafft rejects market share liability for reasons of policy as well: The theory functions to shift the burden to each defendant to exonerate itself as a cause of the injury the plaintiffs claimed. The relinquishment of the "identification [of the defendant] requirement" would mean abandonment of the fundamental concept of law [id. at 247]:

> "the requirement that a plaintiff prove, at a minimum, some nexus between wrongdoing and injury."

Zafft reaffirms the policy that a plaintiff who seeks recovery in tort against one joined as a defendant must identify that defendant as an actor in the production of the harm for which the plaintiff seeks recovery. To require the defendant to exonerate itself as a producer of the harm in very effect is to shift the burden of proof on causation in fact to the defendant, a choice of policy Zafft was not ready to adopt. Zafft rejects market share liability for other reasons of policy as well: The theory functions to allow recovery against a defendant named, but

not identified as an actor in the production of the harm for which the plaintiff seeks recovery, and allows escape from liability only to the defendant who exonerates itself as a producer of the harm. To require a defendant to exonerate itself in very effect is to shift the burden on causation in fact to the defendant, a choice of policy Zafft was not ready to adopt. The court chose to keep intact that "so fundamental a concept of tort law" that [id. at 247]:

> "a plaintiff prove, at a minimum, some nexus between wrongdoing and injury."

Here, the plaintiffs do not ask to be relieved of the obligation to prove "some nexus between wrongdoing and injury." They do not seek that Alcolac carry their burden of proof on the causation element of the cause of action, nor recovery against a defendant not identified as an actor in the production of the harm they claim to their persons and property. They ask only that we accept their evidence - the only evidence available to them - under usual principles of circumstantial proof. Prosser and Keeton, on The Law of Torts § 41 (5th ed. 1984). Here, also [as our fuller discussion shows] Alcolac was not only an identified tortfeasor, but the only reasonably possible point source of the pollution to which the plaintiffs were exposed - hence Alcolac was the only reasonably possible tortfeasor of the injury the plaintiffs claim. Zafft does not bear on the validity of the evidence the plaintiffs adduced to prove these components of the cause in fact element of the negligence cause of action against Alcolac.

The accession by Alcolac medical examiners Emmett and Kerby that these episodes of chemical exposure were the cause of the recurrent [even chronic] dermal, eye and upper respiratory ailments of the plaintiffs was conditioned by the qualification that no alternative point source could reasonably account for the toxic emissions which induced those acute manifestations.

A number of manufacturing and other plants were clustered in the Sedalia industrial area in some proximity to Alcolac. Missouri Pacific Railroad yards, Missouri Pressed Metals, DeLong Welding, Quality Fiberglass [where TV satellite dishes were manufactured] were the most prominent among them. Nikkila, staff director of air pollution control of the Missouri Division of Environmental Quality, Department of Natural Resources, identified the Missouri Pacific Railroad, DeLong and Quality Fiberglass as other point sources of emissions in the vicinity of Alcolac. He acknowledged that at no time during the past seven years was any complaint ever made as to any off-premises odors from any of these sources, nor from Missouri Pressed Metals. The complaints were of odors and emissions from Alcolac, and they were persistent and recurrent. They were complaints also of eye and skin irritations from these exposures. They came from the Elams, Landons, Lawrences, as well as from other citizens. It was the uniform testimony of the public officials as well that there had been no citizen complaints of odors or emissions or physical complaints from the operation of plants in the vicinity other than Alcolac - such as the Missouri Pacific Railroad yard, Missouri Pressed Metal and DeLong Welding. A very few of the witnesses noticed an occasional odor from Quality Fiberglass and other operations, but they were fleeting and of a different quality than those from Alcolac. There was no testimony from any witness - whether presented by the plaintiffs or defendant - that any off-premises odor from any source other than Alcolac ever brought on physical complaint or manifestation. It was the testimony of the witnesses, litigant and nonlitigant alike, that their symptoms, complaints came on after the Alcolac operations began in May of 1978, and that the eye, nose, throat and skin irritations were sequent upon the exposures to the odors and emissions.

The only point sources of emissions in the vicinity of the plaintiffs which used chemicals of some toxicity, or those which could induce any of their physical manifestations were Missouri Pressed Metals and Missouri Pacific Railroad. Missouri Pressed Metals used a solution of trichloroethane to remove grease from the bearings and bushings manufactured there. Trichloroethane evaporates very quickly and gives off the odor of alcohol. It has a very low toxicity but causes irritation to the eyes and irritation to the skin - symptoms many of the plaintiffs exhibited. The vapors of that chemical compound were vented into the air during the cleansing process. The residue was formerly collected in a drum, and then spread on the plant grounds as a weed-killer - a method of disposal the advisory from the manufacturer, Dow Chemical Company, deems safe. The residue is not transported away. These methods of venting of the vapors and occasional disposal have never been cited by OSHA, on the inspections, or DNR. There has never been complaint of any alcohol odor from any source, or that anyone has experienced any symptom or manifestation from any such exposure.[60] Missouri Pacific Railroad, as our narrative already explains, uses a thinner composed of 66 percent toluene in the sign shop operation of that facility. The thinner was identified by DNR as potentially hazardous because of the toluene content. Some 6,336 pounds of that compound are used per year, or about one-third of the annual use by Alcolac.

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[60]Three of the plaintiffs were employed at Missouri Pressed Metals Company during some of the period they claim harmful exposure to the Alcolac chemicals. Linda Sanders operated a machine there for three months in 1980. Joyce Pryor operated a machine for seven months until June, 1979. Joyce Sommers worked machines for two and one-half years. None of them worked with the trichloroethane solvent or were exposed to its vapors. None complained of or experienced irritation or ailment from that labor. In any event, the proviso which conditioned the acceptance as medical fact by examiners Emmett and Kerby of the histories given by the plaintiffs that irritations followed exposure to Alcolac emissions was that no alternative point source of toxic pollution could reasonably account for those acute bodily manifestations.

Missouri Pacific disposed of that toxic waste by deposit into covered pits. It was the virtual unanimous evidence from the litigant and nonlitigant residents in the environs of Alcolac and of these plants - some of whom lived immediately adjacent to them - that there was no annoyance from odors or emissions from these and other industrial sources, but only from Alcolac. It was the actual unanimous evidence that no one experienced any bodily irritation from exposure to any odor or emission from any of these sources, other than Alcolac.

It was the expert opinion of Dr. Carnow that, the evidence just [and previously more fully] recounted assumed as fact, none of the manifestations of the diagnoses entered for the plaintiffs could be attributable to any of these industrial sources as the cause of their bodily disease, except only Alcolac. Nor does the evidence adduced by the defendant tender a basis for inference other than that Alcolac was the only point source of the toxicants to whose exposure the plaintiffs attribute their physical disease and injury. The evidence that the diseases found in the plaintiffs were attributable to Alcolac and not to any alternative source, therefore, was an element of the diagnoses entered by Dr. Carnow, and hence an issue submissible to the jury, and by the verdicts determined in favor of the plaintiffs. We take as proven, therefore, the accession of the Alcolac medical examiners Emmett and Kerby, that the recurrent [and in some cases, chronic] acute manifestations of eye, nose, throat and skin irritations suffered by the plaintiffs were the biological effects of the exposures to the Alcolac emissions. That is to say, we take as proven that Alcolac breached its duty to the plaintiffs to protect them against the risk of injury from harmful toxic emissions, and that the breach of that duty was the cause in fact of the acute biological effects of the recurrent exposures. The plaintiffs are entitled to damages and judgment for those injuries.

## The Issue of Causation in Fact

### The Biological Causation Subelement

The Alcolac contention remains, however, that - the acute bodily manifestations apart - there was no competent evidence that "any act or omission of Alcolac ever caused or was ever linked or connected to any injury to any plaintiff." A plaintiff who sues for injury from exposure to toxicants, of course, must prove not only a demonstrable relationship between the toxicants and biological disease, but also that the disease diagnosed in the plaintiff is consistent with exposure to the toxicants. The relationship between toxicants and biological disease is a subject of toxicology, pharmacology and epidemiology. The diagnosis of disease in the plaintiff consistent with exposure to the toxicants is a subject of medicine, especially occupational and environmental medicine. Sterling v. Velsicol Chemical Corporation, ___ F.2d ___, No. 86-6087, (6th Cir. August 29, 1988) (Westlaw Allfeds Database); B. Black and D. Lilienfeld, Epidemiologic Proof in Toxic Tort Litigation, 52 Fordham L. Rev. 732, 775 (1984).

Alcolac does not argue that the precondition to the proof of biological causation was not met: that the toxicological experts for the plaintiffs, Legator and Carnow, did not demonstrate by valid scientific evidence that the particular contaminants[61] [among the numerous other pollutants emitted] were

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[61]The evidence identified eleven toxic chemicals [among the uncounted others] used and generated in the Alcolac monomer process as of the toxicological properties which cause the kind of biological disease claimed by the plaintiffs: epichlorohydrin, allyl alcohol, ethyl acrylate, toluene, glycidyl ether, cyclohexane, dimethyl sulfate, hydrogen sulfide, methylene chloride, hypochloric acid, and allyl methacrylate. Alcolac argues recurrently that, the toxicological properties of these chemicals notwithstanding, the acknowledgments of toxicologists Legator and Carnow that mere odors do not signify the presence of chemicals means that there was no proof that any of them ever impinged upon the bodily system of any plaintiff, and hence this evidence

capable of inducing the kind of diseases the plaintiffs claim as injury from the chronic exposure. There was agreement among all these experts [Alcolac toxicologist/pharmacologist Schwartz as well] that these chemicals were organ specific as well as systemic: that they all not only attacked target organs, but also had propensity to damage multiple organ systems. There was agreement of their portals of entry into the biological system and that the organs affected were the liver, kidney, respiratory system, central and peripheral nervous systems, and the heart - among others. There was agreement as well that, among the chemicals, epichlorohydrin, dimethyl sulfate and toluene are carcinogens - although

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proves nothing.

There was evidence by witnesses, many of them employees of Alcolac, that many of these chemicals gave off distinctive odors and that those odors were palpable when used or formed in the production process. Warren Fischer, Alcolac plant supervisor, testified that the hydrogen sulfide generated in the bioponds gave off the odor of rotten eggs. That was the testimony of Alcolac witness Nikkila of the DNR, as well as of Dr. Reid of WAPORA. Fischer, and others, described the odor of ethyl acrylate during the production process as that of paint thinner or varnish - another description frequently attributed to the Alcolac odors by both plaintiffs and nonplaintiffs alike. Toluene gave off a sweet odor - also frequently attributed to the emissions during Alcolac production. The acute effect of many of these compounds - allyl methacrylate, allyl alcohol and dimethyl sulfate, among them - was irritating to the eyes, skin, mucous membrane and upper respiratory system. These were effects described by almost all those who were exposed to the odors during Alcolac production - plaintiffs and nonplaintiffs alike. Two of the most dangerous of the chemicals, epichlorohydrin and dimethyl sulfate, have no odor at all.

There was agreement among the experts that all of these chemicals entered into the bodily system by inhalations, as well as by other disparate portals. Alcolac expert Schwartz explained: "When you inhale something you're taking it directly into the circulation." Thus, while there was no direct proof as to which of the Alcolac chemical contaminants any of the plaintiffs ingested at any time [nor could there be in the absence of the records of identification and measurements expected of Alcolac], the coincidence of these odors and the acute manifestations with the emissions from the Alcolac source - and the absence of these odors and manifestations when that source was not active - bears as circumstantial proof that both the presence and concomitant inhalation of those odors were related to the biological disease these chemicals cause, and the plaintiffs claim.

the Alcolac experts considered that not conclusively proven among human populations. [Alcolac expert Schwartz gave opinion, in addition, that methylene chloride was also a carcinogen to humans and glycidyl ether possibly so.] There was agreement also that epichlorohydrin, allyl alcohol, dimethyl sulfate and methylene chloride are mutagens. There was also agreement that epichlorohydrin and dimethyl sulfate, among them, are alkylating agents - compounds which attack protein and kill cells. Chemicals of that ilk, immunologists Zahalsky and Stechschulte agreed, act to alter DNA and to kill new cell formations, and to suppress the immune system. Methylene chloride, Alcolac expert Schwartz added, also acts to suppress the immune system.

Nor does Alcolac on this appeal contend that certain of the organ dysfunction and disease the plaintiffs attribute to the exposure - or the epidemiologic and other scientific sources the experts allude to as ground for opinion - were not of the kind caused by the chemicals emitted by Alcolac. Nor is it disputed on this appeal that the Alcolac chemicals can cause chemical hepatitis, peripheral polyneuropathy, abnormal muscle metabolism and other dysfunctions the plaintiffs relate to the exposure. It was also a matter of acknowledgment by both of the immunologists - Stechschulte for Alcolac and Zahalsky for the plaintiffs - that toxic chemicals of the kind emitted by Alcolac can adversely affect the immune system and that the condition, chemically-induced immune disregulation, may result.

There was disagreement nevertheless between the cadres of experts for the plaintiffs and Alcolac that these chemicals caused the kind of organ disease and dysfunction diagnosed in some of the plaintiffs. The Alcolac experts - toxicologists and physicians alike - did not agree that any of the eleven chemicals identified with the plant production and disposal processes caused biological consequences such as damage to the 8th cranial nerve,

the testicular function in males, or the porphyrin metabolism function of the liver. Thus, they did not agree that the loss of hearing from nerve damage diagnosed in twenty of the thirty-one plaintiffs, or the sperm abnormality diagnosed in all seven of the male plaintiffs who submitted to the analysis or the abnormal porphyrins diagnosed in twenty-seven of the plaintiffs were the kind of biological disease associated with exposure to these chemicals.

The opinions that toluene causes damage to the 8th cranial nerve, that toluene and epichlorohydrin affect the reproductive system, and that epichlorohydrin induces porphyria were rendered into evidence by toxicologists Legator and Carnow. The opinions rested on the results of animal studies related by Legator from the scientific literature.[62] The need for resort to animal studies to determine the effects of chemical toxins - particularly carcinogenic effects - the witness explained, was the long latency period required for biological effects to manifest among human populations exposed to the toxins. The justification for reliance on the test results, Legator explained also, is the metabolic and pathological comparability between humans and the rodent species used for that purpose. Legator concluded: "For the vast majority of chemicals we have to accept animal data and

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[62] It is a rule of our law of evidence, a recognition of necessity, that an expert may corroborate the opinion rendered by reference to standard publications or reported scientific data not otherwise part of the evidence. State v. Greenhaw, 553 S.W.2d 318, 326[11] (Mo. App. 1977); MoBar CLE, Evidence Restated § 703. This rule applies most aptly in mass toxic tort cases where, as here, the determination of the essential cause in fact element of the causes of action depends virtually altogether on scientific testimony. See Federal Rule of Evidence 703; M. Dore, Law of Toxic Torts § 27 (1988).

that has been stated by every regulatory agency, every international body I know of, our ability to get information on compounds that are actually human carcinogens is almost nil."[63] The Alcolac

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[63]The demonstration of a biologically plausible relationship between a chemical toxin and disease is an assessment of risk. The usual methods to measure risk from exposure to chemicals are the epidemiologic studies, animal bioassays and short-term assays [in vitro tests for mutagenic, and hence carcinogenic and other genotoxic, activity of chemicals.] T. Brennan and R. Carter, Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals, 10 Journal of Health Politics, Policy and Law, 33 (1985); Traubman, Statutory Reform of "Toxic Torts"; Relieving Legal, Scientific and Economic Burdens on the Chemical Victim, 7 Harv. Envtl. L. Rev. 177, 186 (1983). The epidemiologic study undertakes to explore and establish a possible association between a factor and a disease between individuals in a population. B. Black and D. Lilienfeld, Epidemiologic Proof in Toxic Tort Litigation, 52 Forham L. Rev. 732, 755 (1984). Since science cannot yet replicate the exact pathway chemicals travel to reach the injured body, nor yet understand the mechanism of cancer, and since these diseases occur at background level even without apparent cause, an epidemiologic study of the whole population at the site of the exposure rather than of the injured individuals yields a more confident correlation between the exposure and the disease. Note, 26 Wm. and Mary L. Rev. 497, 518 (1985). The inferences derived, therefore, are from group-based information, and so are generalized and statistical. Among those inferences may be the attributable risk: the proportion of the disease that is statistically attributable to the exposure - "how much more likely that party is of contracting the disease than he was before the exposure." M. Dore, Law of Toxic Torts § 25.04[2] and [3] (1988). Thus, epidemiology can establish the increased incidence of the disease in a population from the exposure to the chemical toxin, but not whether the exposure caused the particular disease in a particular person. M. Dore, A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-In-Fact, 7 Harv. Envmtl. L. Rev. 429, 431 (1983).

The correlations between exposure [or other factor] and disease derived from an epidemiological study, when deemed statistically significant, suffices in science as an inference of biological causation between the factor and disease - in the sense that "epidemiology allows one to state that a certain factor was the cause of a certain proportion of cases of a given disease in humans." T. Brennan and R. Carter, 10 Journal of Health Politics, Policy and Law at 45; B. Black and D. Lilienfeld, 52 Fordham L. Rev. at 762. The statistical quality of epidemiological correlations notwithstanding, regulators and courts deem such evidence sufficiently significant as to be probative in law as circumstantial proof of biological cause-in-fact in the individual suitor. National Comm'n on Egg Nutrition v. Federal Trade Comm'n, 570 F.2d 157 (7th Cir. 1977) cert.

experts disagreed with the opinions that toluene causes damage to

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denied, 439 U.S. 821 (1978); Reserve Mining Co. v. Environmental Protection Agency, 514 F.2d 492 (8th Cir. 1975); Borel v. Fibreboard Paper Products Corp., 493 F.2d 1076 (5th Cir. 1973) cert. denied, 419 U.S. 869 (1974). See also M. Dore, Law of Toxic Torts § 25.07[1] (1988) and B. Black and D. Lilienfeld, 52 Fordham L. Rev. at 769. The reasons for that acceptance in toxic tort litigation are the refinement of epidemiology as a scientific technique to estimate the proportion of the risk of a disease to exposure to a harmful substance, and the want of better alternative. K. Hall and E. Silbergeld, 7 Harv. Envtl. L. Rev. at 445.

There were no epidemiological correlations in evidence, however, between exposure to the Alcolac chemicals and the risk of damage or disease to the 8th cranial nerve, the male reproductive function, or of the porphyrin metabolism function of the liver - biological conditions diagnosed in numerous of the plaintiffs. The biological cause in fact of those diseases was proven by reference by the medical expert to animal bioassay results. [The sufficiency or competence of that evidence, as we note, to prove biological cause in fact is not a matter Alcolac presents on appeal.] There was no epidemiological evidence adduced to bear on the correlation between exposure to each of those chemicals and the bodily conditions the plaintiffs attribute to the exposure simply because no such study has been conducted. An epidemiological study, the experts testified, is an undertaking usually conducted by governments and foundations, and of a cost few private litigants can afford. [The estimated cost of such a study of the Sedalia population - even if otherwise possible - was one million dollars.] In the absence of direct experimentation on humans with a sample size large enough to yield statistically significant results [a cause precluded by ethical considerations], the epidemiological technique can quantify the hazard of exposure only after a significant number of diseases are incurred by those exposed - and, in the case of increased risk of cancer diagnosed for each plaintiff here - only after a significant number of deaths have occurred. [It is not only coincidence that by 1983 the National Toxicology Program listed only 22 substances as carcinogens - although 95 more are identified "for which there exists substantial evidence of carcinogenesis." T. Brennan and R. Carter, 10 Journal of Health Politics, Policy and Law at 34.] The lengthy latency period for cancer considered, moreover, assures that any quantified assessment of risk, and hence access to epidemiological evidence for proof of biological cause in fact and recovery, is deferred or precluded altogether to a plaintiff. Wagner, Trans-Science in Torts, 96 Yale L.J. 428, 429 (1988).

Animal studies are now also "a widely accepted scientific tool for predicting the effects of chemicals on humans." K. Hall and E. Silbergeld, 7 Harv. Envmtl. L. Rev. at 442; Interagency Regulatory Liaison Group [IRLG], Cancer Policy, 44 Fed. Reg. 39,858, 39,862 (1979). The scientific validity of animal [mostly rodent] tests as a predictive technique rests on the assumption that chemicals behave similarly in different species. The le-

the 8th cranial nerve, that toluene and epichlorohydrin affect the

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gitimacy of that inference of species-to-species extrapolation rests on the fact "that fundamental life processes in mammals and other animals are basically the same as humans" and on the confirmations of a long course extrapolations in biomedical investigations. S. Epstein, The Politics of Cancer, 57 (1978); W. Wagner, 96 Yale L.J. at 432, 442, 444. It has been established, for instance, that animal and human responses are qualitatively similar with respect to all human carcinogens, except arsenic -and "arsenic has recently been reported to produce carcinomas of the respiratory tract in hamsters." Chemical Carcinogens: A Review of the Science and its Associated Principles, 50 Fed. Reg. 10371, 10411 (1985). Scientists now assume, and are expected to assume as a practical matter, that animal models of carcinogenesis are always applicable to humans [id. at 10411]:

> [A] finding of carcinogenicity in rodents is proof that the chemical is carcinogenic in a mammalian species. And "in the absence of adequate data on humans, it is reasonable, for practical purposes, to regard chemicals for which there is sufficient evidence of carcinogenicity in animals as if they presented a carcinogenic risk to humans."

That is also the judgment of the International Agency for Research on Cancer [IARC]. Id. at 10419, n.7. The prudence of that assumption is accepted by OSHA as well. See U.S. Occupational Safety and Health Administration, Identification, Classification and Regulation, pp. 54148-54195.

It is clear that no scientific evidence can speak directly to individual [biological] cause in fact in a toxic tort case. What is available are the extrapolations from epidemiologic, animal and in vitro studies. These are a matter of science and application of sound professional techniques. The relevancy of these extrapolations to attribute disease and biological cause in fact in individual cases is a matter of law. It is evident that scientists assume a significant risk to humans from exposure to chemicals which engender disease and cancer in animals. Animal studies are used and accepted by regulators. See S. Gold, 96 Yale L.J. at 394. It comports well with the substantial factor rule of causation our law applies to such cases [Giles v. Moundridge Milling Co., 173 S.W.2d at 750[6-8]; Restatement (Second) of Torts § 431] to accept the relevancy of "many types of evidence - animal and in vitro experiments, epidemiological data, analogous medical cases - with fact-finders free to decide which of the many inferences urged on them are reasonable." Id. That evidence would weigh with proof of substantial exposure to the chemical toxins and injury consistent with exposure to the chemicals. Such test evidence is all the more probative where - as here - the claim is not for increased risk of organ injury, but for injury actual and subsistent.

male reproductive system, and that epichlorohydrin induces porphyria, not for any reason of animal test invalidity, but because the dosages administered were at "very high levels," and so the resultant pathology could not be extrapolated to humans exposed to chronic, but low level, concentrations.

These opinions were received in evidence. There is no contention by Alcolac on its appeal, or in response to the cross-appeal of the plaintiffs, that these opinions are incompetent to prove that the Alcolac chemicals are not capable of inducing the kind of disease the plaintiffs claim as injury from the chronic exposure - or, for that matter, even incompetent to prove that they were the biological cause in fact of those diseases diagnosed in the plaintiffs.[64] Alcolac argues, rather, that the diagnosis attributes to each plaintiff, chronic systemic chemical intoxication - a disease medicine does not recognize - and the increased risk of cancer - a disease our law does not compensate. In any event, Alcolac argues, the diagnoses were derived by a method pseudo-scientific at best and calculated to mislead. These, however, are contentions of trial error, and raised as such by Alcolac. They do not impinge on the merits of the judgment notwithstanding the verdicts.

4.

Disposition of the Motion for Judgment Notwithstanding the Verdicts

The evidence of causation - as well as of breach of duty and biological damage - was sufficient to prove prima facie the

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[64]The abdominal cancer diagnosed in Mary Landon - and from which she died - was also attributed to the exposure to the Alcolac chemicals. Alcolac makes no specific contention or argument that the cause in fact between the exposure, chemicals, and cancer biological end point of the exposure was not proven by sufficiently probative evidence.

negligence causes of action against Alcolac. The motions for judgment notwithstanding the verdicts in the negligence causes of action were properly denied by the trial court.

B.

Motion for New Trial

Alcolac argues also that if negligence was submissible, then there was error in the admission of expert opinion evidence, and witness evidence, in the formulations of the instructions, the comportment of the trial judge, and other events which prejudiced the defendant and require a new trial. These points and expositions are diffuse, and we confine our responses so as to avoid the repetition the presentation invites.

1.

Expert Opinion Evidence

Alcolac contends that the diagnosis entered for each plaintiff by Dr. Carnow is for a disease medicine does not recognize and otherwise derived by an unscientific methodology. Alcolac describes the diagnosis as: chronic systemic chemical intoxication - and, indeed, that term does not appear in the compiled International Classification of Disease.[65] That term was not tendered as a definitive diagnosis however, but as a generic designation of convenience, rendered into a specific diagnosis by the particular manifestations of bodily disease and dysfunction of the individual plaintiff. The term collapses: "chronic systemic chemical intoxication caused by epichlorohydrin, toluene [and the

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[65]The full term each introduction to diagnosis employs is:
Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including: etc.
Neither does this term, as expanded, appear in the International Classification of Disease.

other identified Alcolac chemicals as impinged upon by the synergistic and pyrolysis effects] with damage to multiple organ systems, including [the listed manifestations of bodily disease or dysfunction of the individual plaintiff]." [See Appendix M-1, M-2, M-3] The medical literature does recognize poisoning from each of the identified Alcolac chemicals. The opinions of all of the scientific experts - toxicologists, pharmacologists, immunologists, environmental medicine physicians and internists -agree that the Alcolac chemical toxins affected multiple organ systems. The liver, kidneys, central nervous system, and peripheral nervous systems were among the bodily functions assaulted by these chemicals. The immune system was also a target - particularly of those toxins with an alkylating quality. The monomer process, Dr. Carnow explained, requires chemicals "which are very active in terms of their ability to destroy and damage and coagulate protein, which is what tissue is." The assessment that these chemicals function as systemic poisons and act avidly on tissue was an opinion shared by Alcolac expert Emmett. There was evidence also that the identified chemicals are metabolic poisons, and impede the activation function of the enzymes. The notion of "systemic poisons" and their effect to damage protein, therefore, is not the neologism of an expert - as the argument intimates - but textbook toxicology:

> "Systemic poisons are typically organic compounds that interfere with the enzymatic reactions, oxygen metabolism, or neural functions. They do so by and large, by virtue of chemical resemblances to organic compounds normally found in animal cells, mostly proteins."

Toxic Chemicals, Health, and the Environment, 41 (L. Lave and A. Upton, eds. 1987) There is no contention that the several biological manifestations and dysfunctions ascribed as subdiagnoses of a particular plaintiff do not describe disease. Chemical hepatitis, porphyria, chronic chemical bronchitis, and the others

are all recognized diseases. Even chemically induced immune dysfunction - challenged by the defense in the testimony of immunologist Zahalsky - was acknowledged as a medical diagnosis by Alcolac immunologist Stechschulte. There was ample evidence that chronic exposure to the Alcolac chemicals resulted in a chronic systemic intoxication to multiple organ systems of the plaintiffs, manifested the particular bodily disease and dysfunction described as to each.

2.

Diagnosis and Methodology

The validity of the diagnoses as recognized diseases notwithstanding, Alcolac argues that these medical opinions rest on evidence which was only scientific simulation and hence not trustworthy. Moreover, Alcolac argues, it was evidence so embossed with the authority of the expert as to convey a false aura of reliability and mislead the jury. The evidence to which Alcolac alludes is what it terms the "frequency tables." They include the series of charts marked with the symptoms,[66] physical examination results,[67] laboratory test results,[68] and diagnosis[69] as to each plaintiff. They include also three summary tables: summary of symptoms,[70] summary of abnormal physical findings, [71] and summary of abnormal laboratory tests.[72] These charts are compilations of the results already derived from the interview, physical examination and laboratory test phases of the patient evaluations by Dr. Carnow at CCA. As compilations, the charts readily reflect the coincidence - or frequency of incidence - of symptoms, abnormal

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[66] See Appendix J-1, J-2, J-3.
[67] See Appendix K-1, K-2, K-3.
[68] See Appendix L-1, L-2, L-3.
[69] See Appendix M-1, M-2, M-3.
[70] See Appendix N-1.
[71] See Appendix N-2.
[72] See Appendix N-3.

physical findings and abnormal laboratory test results among the thirty-one plaintiffs. It is to these "frequency tables" that the defendant addresses the contention of unreliable and deceptive evidence.

It is the Alcolac argument that the frequency tables "were the foundation of plaintiffs' cases - used to prove both causation and injury - on which [Dr. Carnow's] entire testimony was based." Since they were derived by a methodology of only feigned scientific validity - the argument continues - both the summary exhibits as well as the medical opinions of diagnosis on which they rest should have been rejected by the trial court.

We respond at the outset that the summary exhibits were not tendered or received as proof of scientific fact or medical opinion, but only as visual aids to the understanding of the data already fully explained by the testimony of Dr. Carnow. The symptoms, abnormal physical findings and abnormal laboratory test results marked on the SYMPTOMS, PHYSICAL FINDINGS and LABORATORY charts of each plaintiff - in each case found by Dr. Carnow to a reasonable medical certainty to be the effects of the exposure to the Alcolac emissions - were then transposed to the SUMMARY OF SYMPTOMS, SUMMARY OF ABNORMAL PHYSICAL FINDINGS, and SUMMARY OF ABNORMAL LABORATORY TESTS charts. The summaries are merely compilations of the individual charts on those subjects. As such, they depict nothing not already fully explained and subserve, if nothing else, a convenient reference for witness testimony and jury understanding. The admission of the summaries as demonstrative evidence is a matter for the discretion of the trial court. The exhibits, which merely compiled data already explicated by witnesses and received in evidence, were relevant on the issue of the

claim of injury and disease from exposure to the Alcolac chemicals and hence were admissible - if for no other reason. Travel v. Burlington N. R.R., 671 S.W.2d 339, 342[1-2] (Mo. App. 1984) cert. denied, 469 U.S. 1159 (1985).

Upon analysis, the subject of complaint is not the summaries as such, but the validity of the data they compile. Alcolac uses the medium of these exhibits to impugn as "unscientific" the method used by Dr. Carnow to attribute symptoms, abnormal physical findings, and abnormal laboratory test results to the individual plaintiffs. It is therefore, in very effect, a complaint that the diagnoses of disease in the plaintiffs [which derive from these findings] were not proper evidence. The argument as expounded assumes that the sum of the found symptoms, abnormal physical findings and abnormal test results is the diagnosis. It is a conclusion contradicted by the testimony of Dr. Carnow who explained with emphasis that whether symptoms, or abnormal physical findings or abnormal laboratory test results connotes disease is a matter of medical diagnosis integrated from that and other relevant data - including host factors such as genetic predisposition, nutrition and mode of life.

Alcolac argues that the entries the summary tables compile fail to consider alternative risk factors such as smoking, alcohol use, age, family [genetic] history - and other confounding variables related to exposure and effect. Nor, the argument continues, do the entries on the charts tell when any abnormality first appeared or for how long it persisted.

These contentions not only challenge the clear import of the evidence and again confuse prediagnosis procedurer with diagnosis itself, but also misunderstand the methods of environmental medicine evaluation. It is the postulate of environmental medicine, Dr. Emmett as well as Dr. Carnow explained, that cause is cognate to diagnosis. It is for that critical reason, that to

derive a reliable profile that a disease is the effect of an environmental cause, both environmental factors as well as the risk variables of the particular person afflicted must be established. The environmental factors, as our discussion explains, encompass the entire pathway from toxic source to biological disease. They describe the gamut of causation from toxic source to exposure, identification of the toxins, the quantities, their toxicity to humans, the portals of entry into the human system and the biological end point of disease. These were shown by the evidence, direct and circumstantial, of the numerous lay and expert witnesses. The risk variables of the particular person afflicted relate to an assessment of whether the disease diagnosed was the result of the toxins or rather of viral origin, or of a genetic predisposition, or other host factor, or to the mode of life. Those factors were meticulously delved at the interview which initiated the examination of each of the plaintiffs at CCA, and then related into the evidence as already described.

Those entries on the individual charts which depended upon the evaluation of the clinician - such as symptoms given on the interview or abnormal physical findings detected on physical examination - were assessed by Dr. Carnow and entered on the charts as caused by Alcolac only after determination [the environmental factors considered[73]] that the symptom or abnormality was, to a reasonable medical certainty, a biologically expected effect of such an exposure to such a toxic agent. In the case of laboratory test results, which reflected objective values, the reference

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[73]Alcolac reinsinuates through the arguments on the motion for new trial that the environmental factors - such as the identity of the toxins, their measurement, the duration of any exposure to them, and so on - were never competently proven, hence any diagnosis or expert opinion which assumes them for effect is also incompetent. We have responded to those variegated contentions fully in the discussion of the motion for judgment notwithstanding the verdicts.

range governed whether the value found was normal or abnormal. A result outside the reference range was entered on the individual chart as an abnormal finding. Whether any symptom or abnormality connoted disease, Dr. Carnow explained, was a matter of medical diagnosis from the integrated data and assessment of the risk variables. It is only after other possible alternative causes of disease were eliminated that Dr. Carnow ascribed a true etiology. Thus, although immunologist Zahalsky determined from the several panels administered to the plaintiffs that they all suffered from systemic, progressive chemical intoxication so as to dampen their immune systems, the clinical diagnosis entered by Dr. Carnow on the integrated data for Kay Turley and John Phillips did not include immune system dysfunction.

It was this process of differential diagnosis that was applied to each plaintiff seriatim to derive that a disease or dysfunction was caused, or not caused, by Alcolac. It was by this process that the risk variables for the damage to the 8th cranial nerve - and consequent loss of hearing - were assessed to differentiate between a condition brought on by exposure to occupational noise and a loss of hearing from toxic exposure. In other instances, since preexistent diabetes impinges on the protein metabolism function of the liver, that factor was taken into account as a variable to eliminate the Alcolac chemicals as the cause of a manifestation of disease based on an abnormal test result of that a function. Hyperglycemia was distinguished from diabetes, on the other hand, to attribute that disease caused by malfunction of lipid metabolism to Alcolac. The synergistic or multiplicative susceptibility to disease of an alcohol or cigarette user exposed to toxins such as epichlorohydrin and the acrylates was also a risk variable taken into account in the process of diagnosis. It was because personal habit, nutrition,

work, genetic and medical history bear so relevantly upon a valid assessment of diagnosis - and hence causation - that this data was so meticulously drawn from each plaintiff-witess.

It is simply disingenuous, therefore, for Alcolac to argue that the diagnoses entered by Dr. Carnow rested on "overly simplistic summaries of complex issues - [w]ith alternative risk factors [e.g., smoking, alcohol, age, medication] not taken into consideration." The diagnoses did not rest on the summary charts at all, but on the data they reflect, as transformed into a medical opinion through the toilsome process of differentiation of risk variables and the art of the clinician. It defeats fair response for Alcolac to insist otherwise.

It is valid to observe, as does Alcolac, that in terms of epidemiologic principle, age and gender are among the most significant variables in the identification and determination of causes of human disease. T.F. Pugh, Epidemiology, at 103 et seq. (1980). It is not valid to argue, however, as does Alcolac, that the frequency tables [actually, the diagnoses] do not take into account the age span among the thirty-one plaintiffs [from Lyle Turley at 9 and Virgil Bradley at 75] or the gender. The data upon which the diagnoses rest as well as the diagnoses actually entered expressly accommodate to the factors of age and gender - where significant. The reference ranges which defined the normal for a given procedure or laboratory test differed for male and female and age - where those factors were significant to the test. The immune panels - as is the universal procedure [except under the K.U. protocol] - qualify the published reference ranges by those factors. The cholesterol test results were defined that way, as were the numerous components of the genitourinary, liver metabolism and other organ function tests. It was for the very reason that the T cell population, as an absolute number, varies with age that immunologist Zahalsky segregated the plaintiffs into

three age groups - the young, the intermediates, and the older -to enable valid analysis of immune system dysfunction. In the derivation of medical diagnosis from the laboratory and other data Dr. Carnow repeatedly accounted for the age and, where relevant, the gender of the plaintiff. Thus, he discounted as not significant to diagnosis of disease in eleven-year-old Amber Cross the elevated alkaline phosphatase test value [usually an indication of enzyme liver metabolism dysfunction] because that was characteristic of the youth growing-process.

Alcolac condemns as "pseudo-scientific flim-flam" not only the summary charts [diagnoses] but the method of reference ranges which allow for only two results - "'normal' and 'abnormal,' ignoring all degrees" and which omits from the data of diagnosis "the time a particular abnormality may have first appeared or for how long it persisted." This argument is full of ambivalence.

Alcolac simply refuses to accept what all of the scientific witnesses - for the plaintiffs and Alcolac alike - understood: a reference range used by a laboratory represents a statistically significant normal derived from tests done on masses of population so that chance is not a likely explanation for a test result which falls within that range. In terms of diagnosis, the medical witnesses - Carnow, Emmett and Kerby - agreed that a test result outside the reference range must be considered an abnormal test value, but does not by that fact necessarily connote disease. Neither Emmett or Kerby found any cause to doubt the integrity of the tests administered to the plaintiffs, or the reference ranges, or the results [except for the first run of prothrombin time], or the need for them. They disputed only the diagnoses. Emmett and Kerby concluded that, as a clinical matter, the test results, however abnormal, did not [except for the acute effects] signify disease - or if they did, the conditions were not

attributable to Alcolac but to other causes. Contrary to the Alcolac argument, also, in the rendition of diagnosis, Dr. Carnow sometimes gave significance to degrees of test values - those, although barely within or without the reference range, where the particular result was a significant departure from the median.

Alcolac argues most ambivalently when it berates Dr. Carnow, on the one hand, for the "[l]arge numbers of medical tests on [the] many people [which] virtually guarantee getting many 'abnormalities'" and, on the other hand, for the lack of test results "as to when the 'abnormalities' first appeared or for how long they persisted." That is to say: there were too many needless tests applied and too few necessary ones neglected. The testimony of the Alcolac medical experts belie those assertions. The laboratory tests administered to the plaintiffs proceeded under a protocol developed by Dr. Carnow to test those organs and organ functions known to be the biological targets of the chemicals used and emitted by Alcolac. The number and kind of laboratory tests administered to a plaintiff were governed by the nature of the symptoms, physical findings and related diagnostic incidents of the particular evaluation. This enabled an informed selection of laboratory tests among the thousands available for the various organs. They ranged in number from 25 for the youths, Amber Cross and Lyle Turley, to 63 for Carl Berry. There was no suggestion or even insinuation that any of these tests were mock procedures, or for any purpose of trumpery. The tests of the liver, an organ targeted by virtually all of the toxins identified in the evidence, were for the most part organ and function specific. They tested the integrity of phases of the metabolism, excretion and synthetic functions of that organ. The tests Alcolac expert Kerby described for the integrity of the liver cells and function were those administered under the CCA protocol. The tests for the hearing, male reproductive, heart, genitourinary and

other functions were those usually administered for purposes of diagnosis. Neither Kerby nor Emmett doubted the validity of the choice of those procedures, or of the laboratories which administered them, or even of the results - except, once again, for the first run of prothrombin time.

It was the evidence that, as a practical matter, the number, kind and frequency of laboratory tests were constrained by considerations of inconvenience and cost. A number of the procedures cause pain - such as the tests for the motor and sensory nerve responses to determine damage to the peripheral nervous system. Another, causes embarrassment - the production of a specimen for sperm analysis. They are also expensive. The cost of an immune panel was $1000. To the cost of the laboratory services must be added the expense of the diverse specialists who conduct the evaluation interviews, collate the symptomology, the neuropsychological tests for central nervous system damage, and the cadre of other clinicians, consultants, scientists and staff - all of whom had a necessary role in the accumulation of the pre-diagnosis data. The extravagant cost to a plaintiff for the laboratory tests, the technical experts and evidence indispensible to the prima facie proof of the involved cause in fact issue in a toxic tort litigation, is a matter of notice and concern for its access to the courts implications. See Note, A Suggested Remedy for Toxic Injury: Class Actions, Epidemiology and Economic Efficiency, 26 Wm. & Mary L. Rev. 497, 515 (1985); Harris, 40 Sw. L.J. at 949 (1986).[74] There is simply no basis in the evidence to

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[74]The proof of causation in fact in a case of exposure to chemical toxins - particularly a chronic exposure - involves, as our discussion relates, probative evidence of the toxic pathway from the source of the pollution to the person of the plaintiff who makes complaint of biological disease as an effect of that exposure. Typically, such proof involves scientific experts - among them, the industrial hygienist, toxicologist, environmental medicine physician, and where the disease manifests only after long latency or where the claim is of significant risk of disease

support the Alcolac contention that the results depicted on the SUMMARY OF ABNORMAL LABORATORY TESTS chart were without reliability or validity because they represent data from too many or too few procedures. The data the chart compiles, rather, were the results of laboratory procedures acknowledged as both necessary and valid for tests of disease to the organs normally affected by exposure to the identified chemicals. Accordingly, the compiled data the summary charts represent were relevant to the issues on trial and the exhibits were properly admitted. Craddock v. Greenberg Mercantile, 297 S.W.2d 541, 547[3-12] (Mo. 1957). Also, Dr. Carnow derived the medical diagnoses by a method scientifically acceptable and by evidence probative and legally valid.

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yet to manifest, an epidemiologist and statistician. These skills are often found in one expert. [Dr. Emmett was presented as an expert in occupational [environmental] medicine and toxicology. Dr. Carnow was presented as an expert in environmental medicine, toxicology and epidemiology.] It has been observed: "The best witness, of course, would be a medical doctor thoroughly trained in epidemiology, because the need to integrate biology, statistics and common sense to draw proper inferences requires as broad a background as possible." B. Black and D. Lilienfeld, 52 Fordham L. Rev. at 776 (1984).

The total cost to the thirty-one plaintiffs of the litigation we review on appeal was more than $900,000. The expenses paid to CCA for the attention to the plaintiffs over the several years until the date of trial - the laboratory tests it sponsored and conducted and for the professional uses of its experts, staff and facilities and off-premises nonstaff consultants and facilities - was $516,311.40. That cost to CCA included environmental, nonmedical, tests as well. For instance, the plaintiffs sponsored three separate tests to attempt to establish the source of their symptoms and complaints. That was at a time before they had access to any information as to the kind and quantity of chemicals used by Alcolac. Three sets of tests were undertaken by industrial hygienist Boelter and a crew of other scientists. The first two measured air and water samples at several plaintiff residences and air samples taken near the Alcolac premises. The third, taken under the auspices of a court order, allowed entry onto the Alcolac premises to test the bioponds. The plant was then not in operation, so air samples would have been futile. The cost of the last test alone was $20,272; another of the tests cost $35,000.

3.
Competency of Medical Opinion
on
"Nonmedical" Causation

Alcolac not only argues that the medical diagnoses entered for the plaintiffs were derived from unreliable data evaluated by an unscientific method, but also that Dr. Carnow was allowed to render opinion "in the non-medical field of liability causation" - an opinion he was not qualified to give. Liability causation, as Alcolac uses the term, means not an opinion that chemicals had injured the plaintiffs, but that "it was defendant's chemicals which had done so." Alcolac acknowledges that Dr. Carnow was presented as qualified in six separate medical fields, and so was competent to give diagnosis as a medical expert that the injuries were caused by chemicals. Alcolac contends, however, that Dr. Carnow was not presented or qualified as a "non-medical expert," and hence his opinion that Alcolac was the source of the pollution to which the plaintiffs were exposed and the cause of the acute and chronic effects their bodily systems manifested was incompetent.

It is clear, however, that Dr. Carnow was received as an expert in toxicology and epidemiology, as well as in several medical fields, and Alcolac has never contended that the witness was not qualified to render opinion on those "non-medical" subjects. Alcolac intimates, nevertheless, that the matter of pollution source was sufficiently one of common experience and sense as to render the opinion of an expert an invasion of the jury function. The determination of pollution source involves not only the science of industrial hygiene, but also of toxicology, meteorology, and even ambient air modeling. The validation of source, moreover [as experts for both litigants agreed], involves the determination of whether the chemicals emitted by the source were of the kind that reasonably explained the physical effects

manifested by those exposed to them. That is a matter of toxicology and pharmacology, and not of common sense and everyday experience. It is a subject with which the ordinary juror is not likely to be conversant, and so one where the opinion of the expert serves to enable the trier of fact to understand the evidence. Wessar v. John Chezik Motors, Inc., 623 S.W.2d 599, 602[1] (Mo. App. 1981). In such a case, it is no valid objection that the expert testifies on the ultimate issue to be decided by the jury, or that such opinion invades the province of the jury. State v. Willis, 706 S.W.2d 265, 267 (Mo. App. 1986).

The essential flaw in the Alcolac argument is that it would separate environmental cause [liability causation] from biological effect [medical causation] in the diagnosis of disease from toxic exposure. It is a delineation which flaunts and even contradicts its own evidence. Alcolac expert Emmett explained that it is the very function of environmental medicine to establish "a causal diagnosis for a bodily state." The end of such diagnosis is to determine "not only what somebody had, but why it is caused." In that inquiry, therefore, cause and diagnosis are inseparable. It is for that reason, as Dr. Carnow agreed, that the environmental factors [such as source of toxic emissions, identity of the toxins, their toxic profiles, etc.] must be validly established. It is the end of environmental medicine to establish cause, and hence diagnosis, to a high degree of probability. Since there may be "hundreds of different causes" for a physical complaint, Dr. Emmett explained, the environmental medicine investigator uses the technique of differential diagnosis to settle on that cause which explains the complaint with satisfactory scientific probability. An alternative source which may also reasonably explain the complaint is also discounted by a like probability to arrive at the true etiology. It was on these principles of environmental medicine that Alcolac expert Emmett

determined - subject to alternative source being discounted - that Alcolac was the cause of the acute effects suffered and manifested by the plaintiffs.

It simply will not do for Alcolac to contend now that an environmental expert may not give opinion of etiology ["liability causation"] as an incident of diagnosis. Alcolac experts Emmett and Kerby openly acknowledged that such was a purpose of their testimonial role as chosen experts for the defendants. In fact, the etiology of the manifestations of bodily disease or dysfunction noted by the Alcolac medical experts on the diagnosis chart for each plaintiff was entered according to cause, without distinction to "medical" or "nonmedical" cause: Alcolac, Another Cause, Pre-existing, Unknown.[75]

Alcolac argues that, the competency of Dr. Carnow to give opinion on "liability causation" assumed, the opinion was guised in the terminology of epidemiology but not faithful to its principles, hence misled the jury and should not have been received. Alcolac alludes once again to the frequency tables and the complaints of the nonplaintiff witnesses.

It is evident from a glance at the Summary of Symptoms chart that the plaintiffs shared many of the symptoms in common. [The chart merely reflects their trial testimony.] All thirty-one of them manifested some nose, eye, skin or upper respiratory irritation or discomfort. Twenty-four among them displayed tingling, cramps, numbness and other symptoms of peripheral neuropathy. This incidence of symptoms was reflected among the nonplaintiff witnesses as well. The results of the laboratory procedures administered to the plaintiffs [but not, of course, to the nonplaintiffs] established a like commonality of abnormal incidence. These abnormal values were related into evidence by

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[75] See Appendix O-1, O-2, O-3.

the laboratory reports of testimony of the expert, and their coincidence among the plaintiffs noted, and sometimes capitulated in a summary chart. Thus, the charts and testimony reflected that seven of the seven males who submitted to fertility analysis displayed at least two abnormal results. The peripheral nervous systems of ten of the thirty-one plaintiffs tested abnormally. Twenty among the thirty-one plaintiffs suffered damage to the 8th cranial nerve and hearing loss. Twenty-seven among them demonstrated some porphyrin abnormality - a value the expert described as "just incredible." The immune panels established that twenty-six among the thirty-one plaintiffs suffered from an immune dysfunction. This unusual commonality reflected even at the more discrete test levels: ten among the twenty-four plaintiffs who presented themselves for the Midwest/Wheeler immune panel displayed significantly elevated HNK-1 [human natural killer cell] values. There was allusion to other such frequencies.

It is not altogether clear what precise objection Alcolac means to lodge against this evidence of commonality or symptoms and abnormal laboratory results. Alcolac argues that the evidence of commonality does not derive from a true epidemiologic study, and hence the coincidences to which it alludes is without validity on the issue of "liability causation." Moreover, the argument continues, even if properly conducted, epidemiology can only determine which factors may be statistically associated with disease occurrence in populations, and not whether "exposure to a specific chemical causes a specific disease in a specific

person."[76] The salient criticism of the Carnow testimony of the incidence of symptoms and abnormal test results is that "he had not fulfilled the foremost principle of epidemiology, i.e., use of a control group." Rather, the argument continues, the information of symptoms and test results was from a biased group - the plaintiffs themselves.[77]

The witness never pretended an epidemiologic study, however, nor proferred any opinion in that guise. His response on cross-examination explains: "[I]t does not fulfill classic epidemiologic principles, nor was it ever intended to." The allusions to common symptoms and laboratory findings, rather, were for the most part responses to hypothetical questions posed to the witness as an environmental medicine expert for opinion on diagnoses - and hence causation. They bore relevantly to determine whether others had been similarly exposed, and hence on the proof of exposure.

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[76]This argument, as others we have noted, is ambivalent. The point on appeal to which we respond relates to the contention of trial error in the admission of the expert opinion of Dr. Carnow on the "non-medical issue of liability causation." [emphasis added] There is no specific point asserted on appeal, here or elsewhere, that any medical opinion of Dr. Carnow improperly attributed to the Alcolac chemicals the "medical causation" of any biological condition of any particular plaintiff.

This recurrent ambivalence in this phase of argument rests on the insistent Alcolac refusal to accept the effect of its own evidence that diagnosis in environmental medicine is inseparable from cause: that is to say, diagnosis subsumes the valid scientific proof of every element of the toxic pathway - from identification of the source of the pollution, to the identification and measurement of the toxic pollutants, to the fact of exposure, to the toxic qualities of the chemical pollutants, to the biological disease that such toxins may be expected to cause, to the determination of such biological disease in the persons of the plaintiffs. To posit diagnosis, the environmental medicine expert assumes as fact [by the usual hypothetical], the proof of these elements of the pathway.

[77]That observation evidently refers to the punctilios of the "cohort" species of prospective epidemiologic studies. See M. Dore, Law of Toxic Torts § 25.02[3][a] (1988); Black and Lilienfeld, Epidemiologic Proof in Toxic Tort Litigation, 52 Fordham L. Rev. 732, 755 (1984).

They also bore relevantly - especially the commonality of abnormal laboratory results - to confirm that the symptoms were not mimics of disease, but of biological conditions usually expected from exposure to the identified toxins. The common symptoms of eye, nose, skin and pulmonary irritation were "markers" of the connection between these acute symptoms and the observed source of the emissions - Alcolac:

> Those all tell us that not only did these people smell something and see something, but that something got to them, because when it got on their skin, it caused damage, and you could see the damage on their skin. When it got into their throats, it caused sore throats and dry throats. When it got into their eyes, it made their eyes burn and they got red. When it got into their noses, they became chronically irritated and some of them had these terrible nosebleeds, especially the young people, because their membranes are more delicate. All of these are markers. It is almost as if you--they carried--there was a stream from the smoke to the skin. These are very important to us in establishing the fact that here were the offending agents and here are the people and that there is a connection between the two....Extraordinarily strong cause and effect.

The evidence of commonality, in a word, was a means to confirm causation by that "high degree of probability" the scientific method imposes on the environmental medicine investigator for valid diagnosis.

The significance of commonality to cause and effect analysis was confirmed by Alcolac toxicologist Schwartz. He observed that where, for instance [as was usual in the disparity of the medical opinion in the diagnosis of the chronic effects of the exposure on the plaintiffs], "I had one physician saying I interpret this result as porphyria, and another physician saying not, then I have a problem as to find out whether the person is injured or sick in that regard. So, that is what I am talking

about, substantiating the effect." In that analysis, although itself the end of the cause and effect inquiry, "commonality is something you use."

In effect, the diagnoses entered by Dr. Emmett, that the cause of the acute effects manifested by the plaintiffs was exposure to toxic emissions from the Alcolac source, rested on a compound of common symptoms and a logical temporal relationship between the manifestation of symptoms and the episodes of exposure to toxins from an identified point source. The diagnoses of Dr. Emmett and Dr. Carnow differed only to the extent that the latter attributed the chronic effects to Alcolac, and in that process of diagnostic investigation attributed relevancy to the commonality of abnormal test results.

We conclude that the opinion on "liability or non-medical causation" rendered by Dr. Carnow neither invaded the province of the jury, nor rested on unreliable scientific simulations, nor was otherwise tainted by allusions to the nonplaintiff complaints of symptoms and other effects of exposure, nor was the opinion "contrary to undisputed facts" - as the point also - almost casually - adds.

4.

Undue Limitation of Cross-examination

The consolidated cases were tried to the jury over three and one-half months. Of the sixty-nine actual trial days, Dr. Bertram Carnow, a principal witness for the plaintiffs, occupied the stand for eleven days. Cross-examination of the witness, first by one Alcolac counsel and then the other, took almost four days. Alcolac contends nevertheless that cross-examination with the purpose to impeach the witness was unduly restricted by the trial court, and was error.

The complaint cites five subjects on which the trial court would not allow cross-examination: (1) the diagnoses entered by the witness in prior environmental litigations (2) the fees received for testimony in prior litigations (3) that the methodology used by the witness to derive diagnosis in this case was rejected as untrustworthy in prior litigations (4) misrepresentation by the witness of the number of times he failed to pass the internal medicine boards (5) the areas in which the witness was deficient when he failed to pass the internal medicine boards.

The first undue restriction on the impeachment of the witness, Alcolac contends, was that the court did not allow cross-examination to show: "That in several other environmental cases, notably in Illinois and West Virginia,[78] Dr. Carnow had without variation given some 250 other plaintiffs the exact same diagnosis given all 31 plaintiffs here: Chronic systemic chemical intoxication."

In fact, cross-examination on that subject was allowed and undertaken, but after some ten pages of transcript colloquy on the subject with the witness, was curtailed because the inquiry was not impeaching, but only repetitious. The purpose of the cross-examination was to elicit from the expert witness that chronic systemic chemical intoxication was a diagnosis, and that such diagnosis was entered not only for the thirty-one plaintiffs on this trial, but also as to the 125 plaintiffs in the Boggess case, the 47 plaintiffs in an Illinois case, and 65 plaintiffs in another Illinois case. Cross-examination probed three times to elicit from the witness that the designation was a diagnosis and a diagnosis invariably entered in every one of these claims for bodily disease from chronic exposure to toxic chemicals. The

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[78]The offer of proof identified two Illinois cases, but not by formal citation. The West Virginia case is Boggess v. Monsanto Co., No. 81-2098-265 (S.D. W.Va. 1985), a federal district court adjudication.

witness responded to these inquiries with the explanation that chronic systemic chemical intoxication was not a diagnosis as such, but merely a generic term used for convenience and pith. He explained that in the Boggess exposure, for instance, the chemicals involved [except for hydrogen sulfide] were different than those to which the plaintiffs here were exposed, and caused different diseases. Those diagnoses were of diseases caused by chronic systemic chemical intoxication by those specific chemicals - dioxin among them. They attack certain organs and cause certain diseases - as do the chemicals to which the plaintiffs here were exposed, and the chemicals to which the plaintiffs in the Illinois cases were exposed. These chemicals share the characteristic of systemic poisons and so, although they cause different diseases, some organs - such as the liver and immune system - are affected by many of them. The proper diagnosis in any such case, his responses reiterated to cross-examination, is chronic systemic chemical intoxication from hydrogen sulfide, dioxin [or other systemic poison or poisons] including [whatever manifestations of bodily disease or dysfunction resulted from the intoxication]. [That was also a subject of testimony already explained in the case in chief.]

The trial court disallowed further cross-examination on that subject only after it became evident that the line of inquiry was ineffectual as impeachment and unproductive to any relevant end. The trial court observed [a fact already established by the testimony of experts Carnow and Emmett alike] that the essence of the environmental medicine specialty is the diagnosis of disease caused by environmental factors - among them, toxic chemicals which are systemic poisons. Thus, as the court commented a prelude to discontinuance of further cross-examination, it is no more a discredit for the environmental medicine expert to acknowledge that he determined in other plaintiffs chronic systemic chemical

intoxication than for an orthopedic expert to acknowledge that he had determined broken bones in other plaintiffs. The court did allow cross-examination to continue to probe whether in the other litigations the witness had found the same manifestations in those plaintiffs as found in these plaintiffs. That examination ensued, and the response was that there were similar findings of liver disease and immune dysfunction among some of them, but he could not give the incidence without consultation of the personal records.

The interest and bias [and hence credibility] of a witness are always relevant in a litigation, and may be shown, subject only to such limitations on the extent of inquiry as the trial judge may impose in the exercise of a sound discretion. Houfburg v. Kansas City Stockyards Co. of Maine, 283 S.W.2d 539, 548 [18-24] (Mo. 1955). The inquiry defendant insists was a test of credibility, and so a matter for the jury, was only repetitious nonrelevance, and so was properly excluded. Brug v. Manufacturers Bank & Trust Company, 461 S.W.2d 269, 276[5,6] (Mo. banc 1970).

Alcolac contends that the fees received by witness Carnow in prior litigations were a proper subject of cross-examination and that the denial of that line of inquiry was error. The point as fully rescripted complains that the court should have allowed Alcolac to elicit on cross-examination that "in the Illinois and West Virginia litigation, he had received more than $2 million, in addition to the some $566,000 he was here paid." The trial court had repeatedly allowed cross-examination of witness Carnow as to the amount CCA received for its services to these plaintiffs. The trial court also allowed cross-examination as to his involvement in other cases, including the days spent in court for testimony, the number of litigations, by whom employed, and on other kindred subjects. The trial court would not, however, allow

questions as to the amount in fees received by CCA and Carnow for services done in other cases - as an irrelevant and collateral inquiry, more prejudicial than probative.

An inquiry which reasonably bears on the issues in litigation or tends to impeach or discredit the witness is proper cross-examination. The pecuniary interest, or bias or prejudice, of a witness can always be shown. Weatherly v. Miskle, 655 S.W.2d 842, 844[3,4] (Mo. App. 1983). So, the court may, in its discretion, allow cross-examination of an expert witness to show pecuniary interest, or bias or prejudice where these factors can affect the value of his testimony. 31 AmJur 2d, Expert and Opinion Evidence § 50 (1967). In the usual course, therefore, a court allows inquiry as to the amount the witness will receive to testify as an expert, but does not allow cross-examination as to compensation in other cases, unless that bears materially on the interest of the witness in the case in litigation. Board of Public Buildings v. GMT Corporation, 580 S.W.2d 519, 531[15-17] (Mo. App. 1979). Where the inquiry into interest and prejudice as proposed and tendered, however, itself augurs prejudice more than probative purpose, the court may deny its exercise. Id. The court may deny its exercise as well where the inquiry as tendered so lacks definition as to involve collateral and irrelevant inquiry. Hurlock v. Park Lane Medical Center, Inc., 709 S.W.2d 872, 876[1-3] (Mo. App. 1985).

When a tender of impeachment cross-examination is refused by the trial court, the party who seeks to pursue the inquiry must define to the trial court the facts proposed to be shown in the inquiry as well as the witness, exhibit or other evidential source for the proof. The mere statements and conclusions of the interrogator are not enough. Kinzel v. West Park Investment Corporation, 330 S.W.2d 792, 795[1-4] (Mo. 1959). If by testimony, then the witness should be placed on the stand and

the questions propounded and the answers given. If by other evidential source, then that proof should be presented to the trial court. In that manner, the trial court is readily enabled to an informed decision as to whether the evidence bears to impeach or tends to collateral subjects and prejudice. The procedure also enables an informed appellate review on the correctness of the trial court decision. State v. Sullivan, 553 S.W.2d 510, 513[1,2] (Mo. App. 1977). In the absence of an objection or request for further definition, however, the offer of proof need not tender the witness proper, but - if otherwise sufficient - may be accepted by the trial court for purposes of ruling. Stapleton v. Griewe, 602 S.W.2d 810, 813 (Mo. App. 1980). Where the purpose of impeachment is to establish the interest - and hence bias - of an expert witness by evidence of the compensation received in other cases for testimony as an expert, the offer of proof must include evidence that in fact the expert received the compensation. That evidence also - to avoid collateral effect - must raise an inference that the payments received by the witness went beyond the fair compensation usual for such services. Annotation, Cross-Examination - "Professional Witness," 39 A.L.R.4th 742, 758 (1985).

Alcolac argues that Dr. Carnow is a "professional witness," and so to show his interest and bias it was legitimate to elicit on cross-examination that he had received $2 million dollars for services as an expert in cases other than this litigation. The offer of proof was the statement of counsel to the court that the witness had received those sums from the plaintiffs in other cases - only informally identified. The plaintiffs objected to the offer of proof as not sufficient in law. And, in fact, the representations of counsel were the entire offer of proof. There was no request to put the witness, there present, on the stand to establish facts for impeachment. Actually, it would

have been a futile gesture since the witness had already explained repeatedly that the sum charged a plaintiff reflected not his fee, but total expense for work done or sponsored by CCA to derive data for a scientifically competent determination of cause and environmental medicine diagnosis. The charge indeed included not only his medical as well as court services, but also those of the cadre of scientists, medical specialists, and other such professionals, as well as for laboratory services, environmental field tests, and for all those other procedures necessary to establish causation in cases of system diseases from exposure to toxic chemicals. Nor, despite their available medical and scientific experts and a meticulous course of pretrial discovery, did the Alcolac offer of proof assert as fact that any charge of any kind to any of the 300 [or so] plaintiffs in those other litigations was unreasonably disproportionate to the service rendered or was so unfair as to evince an inference that the interest of the witness was in the money paid for those opinions rather than for the probity of those opinions.

We cannot but express uneasiness over the implications for fair adjudications that the extraordinary costs of toxic tort litigation imports. It is an inescapable fact, nevertheless, that a toxic tort cause of action, particularly for chronic effects of chronic exposure, requires the evidence of a range of scientific and medical specialists - however expensive their services. Therefore, to tender as evidence of impeachment that a sum of money was paid as recompense for expert services - per se - proves nothing and discredits nothing. The offer of proof was incomplete, unfocused and collateral. It was within the discretion of the trial court to refuse that component of the offer of proof. Lineberry v. Shull, 695 S.W.2d 132, 137 (Mo. App. 1985).

Alcolac complains also that the refusal of the trial court to allow cross-examination to show that the methodology used by the witness to derive diagnosis in this case was rejected as untrustworthy in prior litigations improperly curtailed the right of cross-examination. That contention assumes that the methodology used in the prior litigations was the same as used in this case to derive diagnosis, and assumes also that the methodology used in this case was amiss. We have already concluded that the methodology used by expert Carnow to arrive at diagnosis for each plaintiff here - that of differential diagnosis of risk variables and confounding factors as to each individual plaintiff - was the orthodox methodology of environmental medicine.[79] That testimony was thoroughly probed on cross-examination, and Alcolac does not and cannot complain of want of opportunity for inquiry.

The prior litigations to which the point alludes are two cases: the Boggess case[80] and the Agent Orange case.[81] In fact [as already discussed] Alcolac examined the expert witness concerning his diagnoses in Boggess, but never questioned the methodology by which they were derived. It was an inquiry never attempted, and hence never denied. The complaint Alcolac makes here was as to a trial event never undertaken, never refused, and so never a subject of error.

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[79] We reject, accordingly, the Alcolac contention that the diagnostic procedure used by the expert to arrive at diagnosis introduces a new methodology not generally accepted "in the relevant scientific community" and so infringes Alsbach v. Bader, 700 S.W.2d 823 (Mo. banc 1985) and Frye v. United States, 293 F. 1013 (D.C. Cir. 1923).

[80] Boggess v. Monsanto Company, 81-2098-265 (D.C. S.D. W.Va. 1985).

[81] In re Agent Orange Product Liability Litigation, 597 F. Supp. 740 (E.D.N.Y. 1984) and In re Agent Orange Product Liability Litigation, Anna M. Lilley, 611 F. Supp. 1267 (E.D. N.Y. 1985).

The other case, Agent Orange, was a national class action by servicemen exposed to chemical defoliants, including Agent Orange, during the Vietnam war. They claimed damages for cancers, birth defects and other injuries to themselves and their families from the exposure to the herbicides. These substances contained dioxin, a chemical highly toxic to humans. In addition to the class action, numerous opt-out plaintiffs filed separate suits. Anna Lilley, wife of deceased veteran John Lilley, was among the opt-out plaintiffs. All of the Agent Orange cases were consolidated and came before federal district court judge Weinstein. The judge approved a $280 million settlement for the class action, but denied recovery to the opt-out plaintiffs by summary judgment. The court concluded that their evidence failed as a matter of law to prove any causative connection between exposure to the defoliants and the cancers and other diseases.

In the Lilley case, the proof of causation for the death of John Lilley from leukemia rested essentially on the affidavit of Dr. Carnow. The affidavit expressed the opinion of Dr. Carnow to a reasonable medical certainty that the numerous diseases and fatal cancer of the decedent were caused by exposure to the dioxin poison during service in Vietnam. That opinion assumed as fact that Lilley was extensively exposed to dioxin in Vietnam and that thereafter he was not exposed to any carcinogens or other toxic chemicals. The information on which the expert relied for medical opinion was supplied by Mrs. Lilley and Comeaux [who served with Lilley in Vietnam], by medical records and scientific studies on exposure to dioxin. The affidavits of Mrs. Lilley and Comeaux to the defendant chemical companies, however, suggested that the service Lilley performed exposed him to other toxic chemicals and that, also contrary to an assumption of the Carnow affidavit, Lilley did smoke. Judge Weinstein rejected the Carnow opinion of causation because, in effect, the information on which the opinion

rested was "sketchy and unreliable." That court disagreed also that the scientific studies "establish[ed] any cause and effect relationship." 611 F. Supp. at 1272. In exclusion of the affidavit as without probative effect on the issue of summary judgment, Judge Weinstein invoked Rule 703 of the Federal Rules of Evidence, which qualifies the date on which an expert may rely for opinion. The court noted that "Dr. Carnow has never examined John Lilley [but] instead relies almost exclusively on hearsay information about Mr. Lilley's symptoms, personal habits and medical background [from] [t]he confused recollection of Mrs. Lilley." Id. at 1280.[82]

The data on which the diagnoses of the plaintiffs here rest, on the other hand, derive from just such procedures of personal interview, description of symptoms and medical history which Judge Weinstein determined an expert in environmental medicine would reasonably rely on for diagnosis, but lacked. Not only were these factors meticulously probated [as our opinion laboriously reports], but also the occupational, genetic and domestic factors which impinged as risk variables in the derivation of

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[82]Of course, John Lilley was already dead when Dr. Carnow was called on to derive an opinion as to the cause of the disease which led to death, and hence an examination was not possible. There is no intimation that the expert had any reason to doubt the information given him by the wife and Comeaux on which the opinion in the affidavit relies, or that he was not qualified to give opinion. Rather, the court expressly accepts the witness as an expert in occupational and environmental medicine. The sufficiency of the opinion of causation entered by Dr. Carnow, even on the data available to him, has been deemed sufficient as a clinical diagnosis of environmental disease by respected comment. See, e.g., Nesson, Agent Orange Meets the Blue Eus: Factfinding at the Frontier of Knowledge, 66 B.U.L. Rev. 521, 526 (1986). The management techniques used by Judge Weinstein as to the Lilley and the other opt-out plaintiffs as in these and other adjudications of the monumental Agent Orange action have been at once roundly praised and roundly condemned. Nesson, 66 B.U.L. Rev. 521 (1986); Cohen, Class Actions, Toxic Torts, and Legal Rules, 67 B.U.L. Rev. 581 (1987); Steinhardt, Mass Toxic Torts May be Hazardous to Your Courts, 20 U.C. Davis L. Rev.657 (1987); Gold, 96 Yale L.J. 376, 392 (1986).

cause and disease by the method of differential diagnosis. There were also the laboratory test results of the numerous organs usually affected by chronic exposure to the toxic chemicals Alcolac used.

The trial court here refused the offer of proof into evidence of the memorandum, order and judgment entered by Judge Weinstein in the Lilley case as impeachment of the validity of the methodology used by Dr. Carnow in the derivation of the diagnoses of these plaintiffs. The court denied the proffer of evidence with the reason:

> There is no relevance to this case at all to what this doctor may have done in another case and other circumstances and one in which, I feel, having read that opinion at length, is so disparate and so contrasting to what we have here today that there is just no basis of comparison, and I just feel it is immaterial, it is irrelevant and should not even be inquired into at any time.

There was no abuse of discretion for the court to conclude that the tendency of the exhibit and any inquiry under it was, not to impeach or discredit, but to get before the jury evidence merely irrelevant and collateral. There was neither error nor prejudice to the defendant. Brug v. Manufacturers Bank & Trust Company, 461 S.W.2d 269, 276 (Mo. banc 1970).

The final contentions on this point relate to two other denials of cross-examination of witness Carnow for purposes of impeachment. Carnow acknowledged that although he was board-qualified, he had never passed the internal medicine board, and in fact had failed them some five times. Those episodes were more than twenty years ago, and he explained the exigencies that accounted for these failures. Apparently, from fault of memory or purpose, the witness had responded to cross-examination in several prior litigations that he had failed the internal medicine boards once or twice, rather than the actual five times. Alcolac offered

to prove these earlier responses as untruths and hence as impeachment of his credibility. The trial court allowed Alcolac to cross-examine as to how many times the witness had failed the internal medicine boards, the scores on those examinations, and how those past failures impinged on his expert opinions where they related to internal medicine. The court would not allow cross-examination to elicit that in prior litigations he had minimized the number of times he had failed those boards and hence misrepresented that fact.

The proposed inquiry was clearly as to a collateral matter, and was properly denied. Hurlock v. Park Lane Medical Center, Inc., 709 S.W.2d at 877[3]. The witness was presented, received and gave opinion, not as a physician expert in internal medicine, but as a physician expert in occupational and environmental medicine. As such, he was competent and qualified to render diagnoses and medical opinions as to the several plaintiffs. See In re "Agent Orange" Product Liability Litigation [Anna M. Lilley], 611 F. Supp. at 1280[5]. In fact, he was board certified and expert as a preventive medicine physician as well as a pulmonary medicine physician - itself a subspeciality of internal medicine. Whether or not the witness forgot, or even lied, as to the number of times he failed the internal medicine boards, therefore, does not impinge upon the credibility of his medical opinions in the case. Id. On the other hand, it was legitimate to cross-examine the witness as to how internal medicine impinged on his diagnoses and to impeach those opinions by his nonexpert status in that field. That inquiry was allowed.

On the occasion of his failed examination in year 1962, Dr. Carnow made inquiry of the American College of Internal Medicine as to "what areas [he] should bone up on," and the response was infectious diseases and some other areas. Alcolac complains

that the denial of the offer of proof to impeach the expert witness by the contents of the letter of response was error. The court excluded the proposed evidence because of remoteness in time - and hence lack of relevance. It is evident that in that twenty-three year interim the witness had developed expertise in at least one phase of internal medicine - pulmonary medicine - and, in any event he was neither presented nor gave opinion as an expert in internal medicine as such. There was no abuse of discretion in the disallowance of cross-examination on that subject.

5.

Instruction No. 9

The negligence count of the petition was submitted by verdict director Instruction No. 9:

> Your verdict must be for plaintiff Betty Elam if you believe:
>
> First, either:
>
> defendant Alcolac, Inc. loaded the bio-oxidation ponds beyond their design capacity, or
>
> defendant Alcolac, Inc. allowed unfiltered toxic waste to go directly into the bio-oxidation ponds, or
>
> defendant Alcolac, Inc. failed to operate the liquid incinerator in a manner which would prevent toxic chemicals from being discharged into the atmosphere, or
>
> defendant Alcolac, Inc. failed to store toxic chemicals so as to prevent toxic chemicals from escaping into the atmosphere, and
>
> Second, defendant Alcolac, Inc. in any one or more of the respects submitted in paragraph First, was thereby negligent, and
>
> Third, such negligence directly caused or directly contributed to cause damage to plaintiff Betty Elam.[83]

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[83]Instruction No. 9 is the paradigm of the submission repeated as to each of the thirty-one plaintiffs. It is formulated in terms

Alcolac contends that Instruction No. 9 prejudicially deviated in form and substance from MAI and that the causation element of the submission otherwise lacked support in the evidence.

Alcolac argues that Rule 70.02(a) enjoins that instructions submit only ultimate issues to the jury, and not detailed evidentiary facts, but that Instruction No. 9 violates that prescription. The argument defines the ultimate issue for submission as: "whether Alcolac permitted chemicals to escape from its premises and come into contact with plaintiffs." Paragraph First of the instruction, Alcolac argues, submits four evidentiary details, none of which passes on the ultimate issue. Alcolac disserves itself with that argument. Paragraph First submits only the conduct which constitutes the breach of duty, and not an issue of causation. To accept the definition of the ultimate issue as Alcolac proposes: "whether Alcolac permitted chemicals to escape from its premises and come into contact with plaintiffs," would leave nothing for the jury to decide because the Alcolac evidence acknowledges that Alcolac was not only the source of the toxic emissions which impinged on the plaintiffs but also that the emissions produced the acute effects to their bodily systems. That is to say, that as to the acute effects, the Alcolac evidence acknowledges both the ultimate issue [as posed by the defendant] that Alcolac not only permitted chemicals to escape from its premises and come into contact with plaintiffs, but also caused biological damage. Yet, there remained another and more serious issue: whether the emissions were of the kind, quantity and duration to expose the plaintiffs to the risk of the kind of

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of Betty Elam because her cause of action for personal injury was the first in the series of the thirty-one submissions. Thus, the contentions Alcolac makes against Instruction No. 9 obtain as well to the other verdict directors under the negligence count.

chronic and latent disease their evidence shows they incurred, and that the chronic exposure actually caused the biological disease diagnosed in the plaintiffs. That issue, however, was not a matter of breach of duty, but of risk assessment and biological causation.

Instruction No. 9, a disjunctive negligence submission modified to apply to joint tort-feasors, is a composite of MAI 17.02 and MAI 19.01.[84] The MAI 17.00 series appertains to motor vehicle negligence. There is no MAI paradigm for the submission of toxic tort negligence. Actionable negligence nevertheless is proven by the existence of a legal duty, failure to perform that duty, and injury caused by the neglect. Hoover's Dairy, Inc. v. Mid-America Dairymen, 700 S.W.2d 426, 431[1,2] (Mo. banc 1985). MAI 17.02 articulates those essential elements of the negligence cause of action and suffices as the construct for the submission of the toxic tort the petitions plead and the evidence proves. A modification of that form was apt for the submission of each of the negligence causes of action proven by the plaintiffs.[85]

Where an MAI must be modified or an applicable MAI instruction lacks, then Rule 72.02(e) requires that the modification or devised instruction be simple, brief, impartial, and free from argument or detailed evidentiary findings. Hall v. County of New Madrid, 645 S.W.2d 149, 151[4,5] (Mo. App. 1982). It is the error in factfinding, in terms of undue emphasis, confusion and

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[84]MAI 17.02 [1980 Revision] Verdict Directing - Multiple Negligent Acts Submitted.
MAI 19.01 [1981 Revision] Verdict Directing Modification - Joint Tort-Feasors.

[85]We do not say that the verdict director [Instruction No. 9] as devised is definitive in the kind of toxic tort cause of action the evidence proves. We respond only to the contentions as presented by Alcolac and determine, for the reasons given, that whatever the evidentiary detail, Paragraph First did not unduly favor the plaintiffs nor tend to confuse or otherwise mislead the factfinder.

other unfairness engendered by unnecessary details, that Rule 72.02(e) means to avoid. Id. Alcolac does not explain how any of these disjunctives of Paragraph First unduly encumbers the fact-finder, or misleads, or clouds the role of the jury to come to a fair determination on a fair definition of the issues. The argument is only that none of these four disjunctive assignments "require[s] any finding of emissions of toxic chemicals actually escaping into the air so as to somehow leave the premises and reach plaintiffs."[86] It suffices to respond again that the submission of the issue Alcolac argues Paragraph First of Instruction No. 9 lacks was not in genuine dispute and was not necessary for proper instruction. Johnson v. Pacific Intermountain Exp. Co. 662 S.W.2d 237, 245[12,15] (Mo. banc 1983), cert denied, 466 U.S. 973 (19 ). There was abundant testimony from lay and expert witnesses that the operation of the liquid incinerator generated a haze in the atmosphere which caused annoyance and symptoms to the residents - numerous of the plaintiffs among them. It was in

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[86] An instruction in that very form was tendered by the plaintiffs at the submission conference but was objected to by Alcolac because "[i]t does not track the usual negligence instruction." That instruction, No. 9A, as originally formulated, tendered on behalf of Betty Elam and the other thirty plaintiffs, submitted [as paragraph Second] the very issue Alcolac now argues was essential but Instruction No. 9 lacks. Instruction No. 9A, however, was withdrawn in the face of the Alcolac objection to the inclusion of paragraph Second and Instruction No. 9 issued instead - as conformed to delete the finding that "Alcolac thereby caused toxic chemicals to escape from Alcolac's premises and to contact and poison plaintiff[s]."

A direct response to the contention on appeal would entail, not whether or not Instruction No. 9 as formulated conforms to the theory of MAI that a jury receives only ultimate issues for resolution, but whether one adversary may, by contradictory objections, create an intractable quandary for the other who in good faith attempts to heed them. It would entail whether MAI and the rationale of Fowler v. Park Corp., 673 S.W.2d 749 (Mo. banc 1984) discountenance a litigant from complaint of error as to a modification his own objection has induced. We have determined that the finding and lack of finding, variously, Alcolac contends for was not an issue in genuine dispute, and hence this aspect of the point on appeal is without merit.

response to these complaints of exposure to emissions from the Alcolac source which brought a number of Alcolac plant managers, president Anderson and Boies to the residences of a number of the plaintiffs.

The fourth disjunctive, that Alcolac failed to store toxic chemicals so as to prevent the compounds from escape into the atmosphere, was supported by the testimony of employees and Alcolac official entries. Some of the toxic waste from Alcolac production, employees testified, were stored in drums of steel or paper and were simply lined outside the buildings and roadway so that large trucks ran over and crushed them. The waste so exposed then evaporated into the atmosphere. There was evaporation also when the chemicals ate through the drums and leaked. There was also evaporation into the atmosphere when drums exploded from the heat, as they did. To control these environmental practices, the abatement agreement concluded between Alcolac and DNR called for the immediate storage of all drummed waste into one of the designated buildings which were designed to collect odors through the system of activated carbon.

The four disjunctives, each, were supported by substantial evidence and hence were submissible.

Alcolac contends also that Instruction No. 9 is erroneous because paragraph Third allows recovery if the Alcolac negligence "directly caused or directly contributed to cause damage to [the plaintiffs] even though the submission was against only one tort-feasor. Alcolac argues that this modification of MAI 17.02 unduly favors the plaintiffs since it imposes a lesser burden than the "direct result" finding of MAI 17.02 proper. The contention Alcolac makes was not presented as an objection to Instruction No. 9, either to the trial court at submission or on the motion for

new trial after verdict, and so is not preserved for our review. Romines v. Donald Maggi, Inc., 636 S.W.2d 130, 132[2] (Mo. App. 1982).

The point is otherwise without merit. MAI sanctions the 19.01 joint-tort-feasor "directly caused or directly contributed to cause damage to plaintiff" modification whether or not the other tort-feasor is a party. Notes on Use, MAI 19.01. It was the recurrent insistence of Alcolac - punctuated throughout the trial - that the Missouri Pacific Railroad Yards, Missouri Pressed Metals, and other industrial plants near Alcolac - were sources of the pollution the plaintiffs experienced and accounted for their symptoms and diseases. It was also the recurrent insistence of Alcolac that the elimination of these operations of alternative sources of the pollution to which plaintiffs were exposed was an element of the causation in fact in a toxic tort action, and hence was a burden of proof cast on the plaintiffs. The plaintiffs acceded to that burden and, by means of a detailed hypothetical to expert Carnow [already fully related] which posed the evidence of the numerous witnesses as to the operations of the Missouri Pacific Railroad yard and the other several industrial sites in the environs - as to any toxic materials used, the quantity and kind of waste, the method of its disposal, and other relevant data - derived the reasonably certain medical opinion from the expert that none of these alternative sources was the cause of any of the disease diagnosed in any of the plaintiffs. The issue of possible alternative source and tort-feasance, therefore, remained before the jury as an element of the proof of biological causation and diagnosis, and the MAI 19.01 "directly caused or directly contributed to cause" modification was proper.[87]

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[87]There was recurrent, spirited debate during pretrial, actual trial, and the interstices in chambers, as to whether the burden to discount other industrial activities in the environs as alternative sources of the toxins the plaintiffs claimed brought on

## Increased Risk of Cancer

Alcolac argues also that it was error for the trial court to refuse to exclude the testimony and opinion rendered by Dr. Carnow that each of the plaintiffs[88] suffered increased risk of

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their diseases properly rested on the plaintiffs as part of the case in chief, or was a matter of affirmative defense for Alcolac. In the course of a particularly animated cross-examination of Dr. Carnow, the contention between counsel over that point of law erupted before the jury. It is a separate assignment of error by Alcolac, which we answer now, that the request for mistrial at the conclusion of that open debate should have been granted. The denial of mistrial was certainly within the discretion allowed the trial court in its management role, if for no other reason than that there was no objection to the interchange, except only the motion for mistrial. The motion to strike the remarks of plaintiffs' counsel, lodged thereafter, was also denied. The record justifies the sense of the trial court action - that there was a parity of culpability.

As we note, with the announced purpose to avoid concern for error, the plaintiffs undertook the proof of the issue. It was the agreed testimony of experts Emmett and Carnow that it is the very function of environmental diagnosis to establish "a causal diagnosis for a bodily state," an inquiry wherein cause and diagnosis are inseparable. It is a diagnosis, as our discussion explains, which not only must assess the source, identity and biological effects of the toxic emissions to arrive at a true etiology from among numerous possible causes, but also differentiates individual risk variables, host and exogenous factors to settle on that cause which explains the complaint with satisfactory scientific probability. So also, and logically, the environmental medicine investigator takes into account alternative sources to derive a scientifically sound cause and diagnosis. Thus, where, as here, the evidence allows a reasonable inference of alternative sources to explain the injury or disease the plaintiffs claim, the medical opinion - as here - must eliminate the alternative source as a biological cause of the complaint or assign its due etiologic significance. In a case such as this, where the plaintiffs claim chronic effects from toxic chemical exposure [so that the substantial factor test of causation in fact applies] the alternative causation issue is more aptly submitted by the 19.01 modification option: "Third, such negligence either directly caused damage to plaintiff or combined with the . . . to directly cause damage to plaintiff."

[88]The prognosis of increased risk of cancer was entered for all of the plaintiffs except Mary Landon who had, by the time of trial,

cancer. It was an error, moreover, made prejudicial by the manner in which that prognosis was adduced from the witness and exploited before the jury. The contention assumes that the medical opinion amounts to a prognosis of increased risk of cancer. It assumes also that Bennett v. Mallinkrodt, Inc., 698 S.W.2d 854 (Mo. App. 1985), cert denied, 476 U.S. 1176 (1986) settles that our law neither countenances nor compensates increased risk of cancer. We do not understand Bennett to announce the peremptory rule Alcolac asserts for that decision. Our response need not delineate the rationale of Bennett, however, because the prognoses do not forecast increased risk of cancer, and in any event the evidence does not prove the cause of action.

It was the theme of the Carnow testimony, repeated throughout a lengthy course of examination, that all of the plaintiffs were exposed by Alcolac to multiple toxins and carcinogens so that they suffer irreversible damage to their organs and to their immune systems. Since a single molecule of a carcinogen [such as epichlorohydrin] can alter DNA and so induce a deformed cell line - and malignancy, the more the exposure and the more of the chemical, the more the probability of cancer. Since the immune system - the major defense against cancer - has been irreversibly damaged - the chronic exposure heightens the probability of an eventual malignancy. In this exposition, the prognosis and medical opinion entered by Dr. Carnow for each one of the plaintiffs hovered between certain cancer and risk of cancer:

> A. ...a single molecule of epichlorohydrin can cause alterations of a single cell, of its DNA, the protein in the cell. If that cell is not killed by the immune system, and if that cell is growable --sometimes the deformity is such that they won't grow, they die--and, then, conceivably, it can grow, but, of course this is--the more of the

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already contracted the disease. She died during the pendency of appeal. Dr. Carnow described Mary Landon as "the first one [plaintiff] to show her cancer." The expert attributed her cancer to the Alcolac emissions.

chemical and the more repeated the exposure, the more the probability that many cells will be altered, and of these many cells that will be altered, one of them may not be killed by the immune system, and that that cell, then, can go on and develop a new cell line, which could, then, become malignant. That's what it is.

But, there is no question that the more the exposure, and the chemicals that you were exposed to, the more toxic in terms of carcinogenic properties, and the more the intensity of your exposure, the more the probability that you will develop cancer.

Q. And your testimony is that every one of these people, every one of these 32 people, will have cancer before they die?

A. No, I never said that. I said that these people have abnormal immune systems, and that because of that, they are at very high risk to cancer.

The next thing I said was that these people have been exposed to multiple carcinogens, not just one, but multiple carcinogens, and some of these carcinogens are extraordinarily toxic, like epichlorohydrin, and that if these people live long enough, that I believe that they will die from a malignancy.

Now, there is a thing that we call competing risk in epidemiologic parlance, and competing risk means that they may die from something else before they die from the cancer, because the problem we have here is that these people have suffered systemic poisoning, and it may well be that they will die from something else as a result of their exposure, and the breakdown of their body systems, you know, the porphyrins and so on, that they may die from another disease, liver disease, or one of the other diseases before they get the cancer; yes.

Q. Are you prepared to quantify that risk that you are talking about?

A. No, I cannot do that. There is not--there is not enough information in science to do that.

The ambivalence of opinion, between certain cancer and increased risk of cancer, persisted in the course of prognosis of the individual plaintiffs. In the assessment of plaintiff Ethel Berry, the expert explained the "very guarded" prognosis entered:

> And that is why we put this in. And, again, there are many reasons for this. So many systems involved, a lot of liver diseases, heart disease and the very severe immune system dysfunction, which makes this--and I should again note that part of what goes into my making my prognosis is the fact that all of these people are at extraordinary risk to developing cancer for two reasons.

> One, they have been exposed to a host of cancer producing chemicals, some of them direct carcinogens, like epichlorohydrin. That means that they don't need to work with anything else. They can directly destroy and start cancer. And the second reason is because the immune system, which is the major defense against cancer, had been severely damaged in most of these people. [emphasis added]

In the assessment of plaintiff Dorothy Bradley, on the other hand, the expert explained the "very guarded" prognosis entered:

> And, again, one of the components of the very guarded diagnosis [sic] is it's my opinion that if these people live long enough, they will develop cancer because of their exposure to multiple carcinogens and because of the damage done to their immune system.

The arguments of the litigants are indifferent to the variableness of this medical opinion and its implications as valid prognosis. Alcolac argues only that the testimony was inadmissible because our law does not recognize a cause of action for increased risk of cancer. The plaintiffs respond that the opinion - that all of the plaintiffs, if they live long enough, will die from malignancy - because rendered as a reasonable medical certainty, validates that forecast as medical evidence.

A prediction that cancer is certain from exposure to carcinogens, however, is a forecast science does not deliver and an opinion, however clinical, medicine cannot derive. It is a commonplace that science has not yet "penetrated the molecular puzzle of cancer." That is to say, the mechanisms of carcinogenesis are still unknown. Thus "when a scientist states that a chemical causes cancer, the term cause is consciously hedged, and necessarily burdened by some degree of uncertainty." Brennan & Carter, Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals, 10 Journal of Health Politics, Policy and Law 33, 46 (Spring, 1985). And,

"although we accept that the chronic exposure of a number of mammalian tissues to a variety of chemical agents often results in the evolution of the exposed cells to malignancy, there is no phase or facet of this process, from its primary cellular interactions to its terminus in the death of the host, that is fully understood. We cannot state with certainty that the process results solely from the interaction of the suspected chemical agent with target cells." Carcinogens: Identification and Mechanisms of Action, 5 (Griffin & Shaw ed., 1979). And, in fact, the testimony of expert Carnow abounds with recognitions that the cancer mechanism cannot be predicted because "each person, based on their genetic constitution, is at different risks . . . based upon the strength of their immune systems [and] a whole bunch of individual factors."

That the prognosis of certain cancer was articulated in terms of reasonable medical certainty only confounds the sense of that opinion. A medical opinion that a future damage or result will follow is an estimate of probability, and not a declaration of certainty. It informs that the consequence is more likely than not. Crawford v. Chicago-Kansas City Freight Line, Inc., 443 S.W.2d 161, 165-7 (Mo. 1969); Dallas v. Burlington Northern, Inc., 689 P.2d 273, 277[6,7] (Mont. 1984); Lane v. State Farm Mut. Auto Ins. Co., 308 N.W.2d 503, 512[5] (Neb. 1981). A probability that cancer will manifest from tortious conduct is an opinion that the conduct has increased the risk of cancer.

We assume, as does Alcolac, that the forecast by Dr. Carnow of future cancer for the plaintiffs is a prognosis of increased risk of cancer from the chronic exposure. A recovery for increased risk of cancer, however - whatever the theory adopted - involves proof of a quantified risk. One theory treats the cause of action for increased risk of cancer as a present injury with future consequences. It allows recovery if the toxic

exposure has induced some biological manifestation from which the anticipated cancer is reasonably certain to occur - as quantified by expert testimony as a probability of occurrence of greater than fifty percent. If the cause of action is proven, there is a full recovery of damages as for a realized cancer. Jackson v. Johns-Manville Sales Corp., 781 F.2d 394, 411[10] (5th Cir. banc 1986) cert. denied, 478 U.S. 1022 (1986); Sterling v. Velsicol Chem. Corp., F.2d , (6th Cir. No. 86-6087, adopted August 29, 1988) (Westlaw Allfeds Lib.); Brafford v. Susquehanna Corp., 586 F. Supp. 14, 17[3,4] (D. Colo., 1984). See Gale & Goyer, Recovery for Cancerphobia and Increased Risk of Cancer, 15 Cumb. L. Rev. 723, 736 (1985). The other theory treats increased risk of cancer as a present invasion of a legally protected interest, and hence actionable even absent any manifestation of injury. The increased risk, as quantified by expert testimony as a percentage of probability, measures the recovery. If the cause of action is proven, the damages are awarded for a cancer caused by the identified toxic chemicals but proportionately reduced by the quantified probability that the plaintiff will not manifest cancer. See Gale & Goyer, 15 Cumb. L. Rev. at 742; Note, An analysis of the Enhanced Risk Cause of Action (Or How I Learned to Stop Worrying and Love Toxic Waste), 33 Vill. L. Rev. 437, 456 (1988); Note, Increased Risk of Cancer as an Actionable Injury, 18 Ga. L. Rev. 563, 589 (1984); Restatement (Second) of Torts § 7 (1985); Pelcha v. United Amusement Co., 44 Or. App. 675, 606 P.2d 1168 (1980); Jordan v. Bero, 210 S.E.2d 618, 640 (1974) (Neely, J.,concurring).

The proof of the cause of action under either theory, therefore, requires opinion of quantified probability of disease. We agree with Alcolac that the acknowledged inability of Dr. Carnow to quantify the risk rendered the medical opinion that all

of the plaintiffs were at extraordinary risk of cancer nonprobative[89] - as that evidence impinged upon the recovery of damages for increased risk of cancer. The evidence of significant, albeit unquantified, risk of cancer[90] [as well as other disease] from the exposure to the toxic Alcolac shown, however, was competent to prove, as a separate element of damage, the need for medical surveillance of the immune system and other organs, and hence was admissible for that purpose. Hagerty v. L & L Marine Servs., Inc., 788 F.2d 315, 319 (5th Cir. 1986); Ayers v. Jackson Township, 106 N.J. 557, 525 A.2d 287, 315[11] (1987); Askey v. Occidental Chem. Corp., 102 A.D.2d 130, 447 N.Y.S. 2d 242, 247[6] (1984). Indeed the prudence and urgency of such a course for early detection of serious disease from the chronic exposure was

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[89]The opinion of increased risk of cancer - as an omnibus prognosis for all of the plaintiffs - was not probative for another reason. It was not supported by the evidence as to plaintiffs John Phillips and Kay Turley. The opinion of increased risk of cancer rested on two premises: all of the plaintiffs had been exposed to multiple carcinogens and that their immune systems - "which is the major defense against cancer" - was damaged. The DIAGNOSIS chart meticulously devised by the witness for each plaintiff and explained as to each entry was devoid of indication of any immune system disregulation, dysfunction or even abnormality as to plaintiffs John Phillips and Kay Turley.

[90]The unwillingness of Dr. Carnow to quantify the risk of cancer his medical diagnosis attributed for every plaintiff rested on the premise that "there is not enough information in science to do that." An accepted means to quantify risk of disease is by the epidemiologic study. [See footnote 61 and Note, An Analysis of the Enhanced Risk Cause of Action, 33 Vill. L. Rev. 437, 457 (1988)]. We note that in the course of testimony, Dr. Carnow, an expert toxicologist, alluded to an epidemiologic study which "more strongly prove[s]" that exposure to epichlorohydrin presents a risk of cancer to humans. In evidence also was the acknowledgment of Alcolac expert Emmett that recent publications conclude that epichlorohydrin presents a risk of cancer to humans. A medical opinion of increased risk of cancer, albeit rendered quantitatively, nevertheless remains a clinical diagnosis and, as such, properly rests on clinical as well as scientific data. Jackson v. Johns-Manville Sales Corp., 781 F.2d at 411; Sterling v. Velsicol Chem. Corp., F.2d adopted August 29, 1988 (Westlaw Allfeds Lib.).

the agreed advice of both immunologists, Zahalsky and Stechschulte. Also, those among the plaintiffs diagnosed with porphyria and other chronic diseases were advised by CCA to monitor the conditions by appropriate medical oversight. Compensation for necessary medical expenses reasonably certain to be incurred in the future rests on well-accepted legal principle. Wilcox v. Swenson, 324 S.W.2d 664, 673 (Mo. 1959); MAI 4.01. See Note, Medical Surveillance Damages: A Solution to the Inadequate Compensation of Toxic Tort Victims, 63 Ind. L.J. 849 (1988) (authored by Allan T. Slagel)

Alcolac does not complain, however, that the evidence to prove the need for medical surveillance of disease not yet fully realized was improperly received. It argues in effect, rather, that other evidence, directed to prove that all the plaintiffs were destined to manifest cancer from the chemical exposures, was not only nonprobative but altogether collateral to the need for medical surveillance, and hence should have been excluded. There is no doubt from the colloquy of counsel with the court that the evidence to come from Dr. Carnow was the medical prognosis that all of the plaintiffs were at extraordinary [or certain] risk of of cancer, and that such evidence was to prove damages for unquantified increase risk of cancer. Nor is there doubt [as counsel for plaintiffs explained to the court] that those claims were a prominent component of the damages asserted for recovery from Alcolac. In those circumstances it was an abuse of discretion to have received the testimony and opinions. Of course, the trial court is allowed a wide range of discretion to admit or exclude doubtful evidence. Lawson v. Schumaker & Blum Chevrolet, Inc., 687 S.W.2d 947, 951[1,2] (Mo. App. 1985). It is the role of the trial court nevertheless to exclude evidence when the danger of prejudice outweighs its probative benefit. Jones v. Terminal R.R. Ass'n of St. Louis, 242 S.W.2d 473, 477[6-10] (Mo. 1951). The

evidence of increased risk of cancer [by then long since formed without quantification] introduced a collateral issue of recovery which suggested to the jury an improper basis for decision and confused the subject of damages. It was error not to have heeded the Alcolac objection to its admission. Conley v. Kaney, 250 S.W.2d 350, 353[3,4] (Mo. 1952).

Alcolac argues that the evidence of increased risk of cancer was not only erroneous, but inflammatory and prejudicial as well. The extravagant color used by counsel to elicit the medical opinion from the expert suggests just such an effect.

> Q. Yes. And under the subject of time bombs, can I write the word cancer?
>
> A. Yes, indeed.
>
> Q. All right. And the fact that these time bombs will go off in each and every one of my clients' bodies, is that a fact that you are telling this Judge and this jury based on reasonable medical certainty
>
> A. It is my opinion that if these people live long enough, they will each have cancer, yes.
> [emphasis added]

That vivid figure of speech was not only reinsinuated to the jury in the course of an attentuated risk of cancer medical proof, but also on closing statement to argue compensatory and punitive damages:

> He talked about the fact that what is in the bodies of my clients has created time-bombs. And it is a good analogy, because you don't know exactly when they are going to explode. One of our clients has already had her cancer, and she's beginning to show.
>
> . . . .
>
> [T]hey will all in fact surely contract cancer if they live to a normal life expectancy.
> [emphasis added]

The medical opinion of the risk or certainty of cancer, albeit not always in the same tone, was argued to the jury repeatedly to exhort damages - actual and punitive.

We agree that it was error for the trial court to allow the evidence and argument.[91] We defer consideration of how this

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[91]The error we determine as to the evidence and argument of increased risk of cancer is that the cause of action, even if current, was not quantified and could not have been proven, and so those trial events only served to confound the legitimate issues. That error affects all of the plaintiffs, except Mary Landon. There was no claim of increased risk of cancer as to Mary Landon, but of a cancer, actual, diagnosed and treated. The DIAGNOSIS chart devised by Dr. Carnow for Mary Landon includes abdominal cancer as a subdiagnosis. It was the opinion of Dr. Carnow, based on reasonable medical certainty, that the abdominal cancer was caused by the prolonged exposure to Alcolac carcinogens. That opinion rested on the humerous factors of risk assessment which impinge on the determination of generic and biologic causation in fact. That opinion gave a prominent role to the process of cancer induction to the quality of epichlorohydrin as a direct carcinogen. The medical opinion that the Alcolac chemicals caused the abdominal cancer in Mary Landon was derived - as were all the other subdiagnoses noted as to each plaintiff - as a differential diagnosis which took into account personal, occupational, medical factors and risk variables.

Although Alcolac assails the "methodology" by which this and the other diagnoses and medical opinions of Dr. Carnow were derived [arguments we full considered and rejected], there is no contention against the competency of the scientific data on which the medical opinions rest. To be sure, the motion for judgment notwithstanding the verdicts asserts the encompassed contentions that there was no substantial proof that Alcolac ever emitted from the premises any injury-causing substance which any plaintiff ever inhaled or ingested, or that "any act or emission of defendant was ever linked or connected to any injury of the plaintiff." Since it was acknowledged - and in any event under the evidence cannot be disputed - that the acute physical effects suffered by the plaintiffs were attributable to the Alcolac emissions, the point on appeal that there was no cause in fact proven between the Alcolac activity and any biological injury to any plaintiff is not tenable.

The Alcolac appeal, however, does not assert any specific contention that, as to any specific diagnosis of chronic disease the evidence attributes to its operations, the diagnosis was incompetent as unsupported by reliable scientific data. To be sure [see footnote 61, supra], the Alcolac experts did dispute that the diagnoses of disease of the 8th cranial nerve, the male reproductive function, and of the porphyrin metabolism from exposure to the identified Alcolac chemicals were scientifically valid. It is not a matter Alcolac presents on appeal, however, any more than is the diagnosis of abdominal cancer in Mary Landon. Therefore, any such question is not before us. Thummel v. King, 570 S.W.2d 679, 685 et seq. (Mo. banc 1978).

There remains the diversion of medical opinion between Dr. Carnow and the Alcolac experts Emmett and Kerby, as to the etiology of the abdominal cancer in Mary Landon as a matter of differ-

error may have prejudiced the damages and verdicts, however, until treatment of the cross-appeals of the plaintiffs. The verdicts returned actual and punitive damages for each of the plaintiffs on the negligence count, but they were set aside and new trial entered on those issues. The appeals of the plaintiffs seek reinstatement of the verdicts. Thus, the disposition of those appeals impinge on the Alcolac contention that awards of damages were unfairly influenced by the evidence and argument of increased risk of cancer.

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ential diagnosis. The Alcolac experts did not contest that the cancer was a disease that exposure to the Alcolac chemicals could induce. It was the testimony of the Alcolac and plaintiffs' immunologists, toxicologists and physicians alike, that exposure to the Alcolac chemicals could result in the suppression of the immune system. It was the testimony of Alcolac immunologist Stechschulte that a disregulated immune system could result in cancer. He gave opinion that the radiation treatment for the cancer "could certainly have been a possibility" to explain the "clearly abnormal" immune system the tests of Mary Landon revealed. It was the opinion of Dr. Carnow, rendered as a reasonable medical certainty, however, that the suppression of the immune system was a condition which antedated the cancer radiation treatment, and was caused by the exposure to the Alcolac toxins. The suppressed immune system left her bereft of that defense and vulnerable to the effects of the Alcolac carcinogens, to which she then succumbed. The radiation simply reinforced the dysfunction of an immune system already suppressed. It was the opinion of Dr. Emmett, on the other hand, that a genetic cause was "much more strong and likely" than exposure to the Alcolac chemicals: the condition of congenital spherocytic anemia. It is a condition, as Dr. Emmett understood from the family history, which has resulted in leukemia-related cancer in "several people of the family." That basis for etiology diagnosis, Dr. Carnow explained, was mistaken from a misconception harbored by Mary Landon and then related to Dr. Emmett as part of her family history - and by him used as a premise of diagnosis. The medical records confirm, rather, that the family malady was not related to leukemia or any other form of pre-cancer, but was an anomaly of the red blood cells. The anomaly is corrected by a splenectomy - the removal of the spleen. That was the procedure undergone by thirteen Landon family members, and not treatment or concern for leukemia.

The etiology of the abdominal cancer, therefore - whether the exposure to the Alcolac chemicals or the genetic anomaly -was fully before the jury. The question of the biological causation of the abdominal cancer in Mary Landon was not that of the competency of the opinion, but its credibility. The jury resolved the issue in favor of Mary Landon and against Alcolac.

7.

## Miscellaneous Errors

Alcolac raises a cluster of other contentions of error. There is complaint that the court did not manage the trial well, that improper closing argument was allowed, and of still other evidentiary errors. To the extent that these complaints, however guised, reargue evidence and points already considered and determined, they are redundant and will not be addressed.

### a.

### Conduct of the Trial

There is complaint that the court lacked firm control over the proceedings, procrastinated rulings, and erred as to others. There is complaint also as to the order of cross-examination imposed on the defendant, and as to comments used by the court in the course of ruling. A number of these complaints are as to insubstantial events. Others are already answered. As to the rule of cross-examination, the court denied the request of Alcolac to interrupt the direct examination of witness Carnow at the conclusion of his testimony on each of the thirty-one plaintiffs, and ordered, rather, that cross-examination follow at the conclusion of direct as to all of the plaintiffs. Alcolac argues that "[t]he manifest confusion this engendered with the jury is obvious in the verdicts they rendered." It is ironic that Alcolac claims as the source of verdict error the very procedure its insistence imposed on the plaintiffs: the concurrent submission and argument of all thirty-one claims to the jury for verdict. This point is denied - as are the others which complain about the decisions on trial procedure. The rulings the complaints cite are well within the discretion of the court to make in the course of

the management of a trial, and were not otherwise improvident. Holt v. Queen City Loan & Inv., Inc., 377 S.W.2d 393, 400[14] (Mo. 1964).

Alcolac complains also that a comment used by the trial court in the course of ruling was tendentious and unduly unfavorable to the defendant. It argues that the denial of a mistrial was error. Counsel for the plaintiffs used a large tablet on which significant testimony was noted. There was no objection to that procedure. The witness then under examination was Bregman of WAPORA. It had been elicited that the design of the Alcolac bioponds was based on information furnished WAPORA by Bouroff of Alcolac "as to what would be going into the ponds." Counsel for plaintiffs then continued the interrogation and probed for the word he would inscribe on the tablet, as was his practice:

> Counsel: Based on what turned out to be <u>false</u> projection?
>
> Bregman: Highly erroneous projections. I would rather think they were <u>erroneous</u> than false.
>
> Counsel: That is a longer word.
>
> Bregman <u>Mistaken. Mistaken. Wrong.</u>
>
> The Court: Put <u>bad</u>.
>
> Bregman: <u>Bad</u>.
>
> Counsel: That helped me a lot.
>
> The Court: <u>That cuts it right down to where it really hurts</u>.
>
> [emphasis added]

The defendant moved for mistrial, and mistrial was denied. The court immediately addressed the jury:

> Ladies and gentlemen of the jury, the last comment that I made concerning the word "bad" is not a personal observation of mine about the merits of the case. I wanted to get the shortest word possible to put on this chart over here, and

> since erroneous was too long, false gave the wrong picture, it might have indicated deceit, I just took the word bad, and that is what I meant by cutting it down. It is not to hurt anybody in the case. It is the Court's shorthand way of getting something on the board quickly, and you should not interpret that to mean that anybody has done a bad job or that the defendant is right, the plaintiff is wrong or any other way.
>
> . . . .
>
> It was my choice of words and the comments added gratuitously after that, which I am instructing you to disregard.

It is improper for a judge by act, conduct or remark to color the neutral status that role entails. State ex rel. State Highway Comm'n v. Thurman, 427 S.W.2d 777, 781[2,3] (Mo. App. 1968). The comment of the trial court, however hedged by proper purpose, was indecorous and was better left unsaid. In the usual course, counsel should be left to elicit their own testimony from the witnesses. In the circumstances presented, however, the jury could not have but understood from the explanation and instruction given them by the court immediately afterwards that the comment was not meant as an expression of belief or favor of either litigant. State ex rel. Mo. Highways & Transp. Comm'n. v. Legere, 706 S.W.2d 560, 569[13] (Mo. App. 1986). The explanation and instruction cured whatever prejudice may have resulted from the error, and the mistrial was properly denied. St. Louis County v. Seibert, 634 S.W.2d 590, 592 (Mo. App. 1982).

We note as a tribute to the adroitness of the trial judge and the ardent advocacy of counsel that such untoward incidents were remarkably infrequent for a trial so intricate, novel and perdurable.

b.

Improper Closing Argument

Alcolac contends that the argument to the jury concerning the evidence of leaks, chemical spills, bad odors, explosions and other such incidents were beyond the four alternative disjunctives submitted by the negligence verdict director, and hence was improper. MAI enjoins that a verdict director submit only ultimate issues, and leaves the evidentiary detail to argument. Rule 70.02(1); MAI at XXXVI (1981). The verdict director Instruction No. 9, we determined, was a construct conformable to MAI. It is proper for a plaintiff, within an ambit generously drawn, to argue the effect of the evidence on the ultimate issues submitted to the jury. Lewis v. Bucyrus-Erie, Inc., 622 S.W.2d 920, 926 (Mo. banc 1981). The intertwined proof of breach of duty, causation, biological consequence and damage a toxic tort case requires, moreover, justifies a commensurately wide scope of argument, explanation and comment by counsel. It was not an abuse of discretion for the trial court to have allowed the argument Alcolac found objectionable. S.G. Payne & Co. v. Nowack, 465 S.W.2d 17, 20[5,6] (Mo. App. 1971).

Alcolac contends also that counsel for plaintiffs in closing argument attributed testimony to Dr. Allcorn, the Sedalia practitioner who treated a number of the plaintiffs at an early stage, that were not in the record. The contention is without merit. The transcript confirms that the closing remarks were based on evidence already before the jury and so were proper. Davis v. City of Independence, 404 S.W.2d 718, 720[2-4] (Mo. banc 1966).

Alcolac contends also that certain words and phrases elicited from the witnesses and used by the plaintiffs in interrogation and argument were inflammatory and should have been

excluded upon objection. Alcolac cites such terms as "Alcolac victims," "toxic chemicals," "Love Canal," "Bhopal," and others. Some of the terms to which Alcolac ascribed emotional color were altogether innocuous. Some others, such as "toxic chemicals," were part of the legitimate proof and inescapable. Others still were attended by explanations from the court as to how they related to the issues on trial. In the full context of use, none of them were calculated to inflame or prejudice the verdicts of the jury.

We agree nevertheless that one term recurrently used throughout the trial - chemically induced AIDS - was not only gratuitous, but inflammatory and prejudicial. It was agreed by the experts that exposure to the toxic chemicals identified as used and emitted by Alcolac could work to suppress the immune system. The immunologists for both litigants agreed that the condition went by the medical term chemically induced immune disregulation [CIID]. It was the testimony of plaintiffs immunologist Zahalsky that when the chemically induced immune disregulation was characterized by an imbalance betw n the helper and suppressor populations of the T-cells, the condition is more particularly described as chemically induced AIDS. That is because in AIDS the virus works to induce an imbalance between the helper and suppressor populations of the T-cells with the result that the helper cells are functionally wiped out. The difference between the two conditions is that toxic chemicals induce one, and a virus induces the other. Zahalsky found that four among the plaintiffs - Virgil Bradley, Dorothy Bradley, Malva Gehlken and Dainie Landon - displayed the chemically induced AIDS condition. Eight others will develop that condition "somewhere down the line."

There was no evidence that any plaintiff suffered from viral AIDS, nor was that a claim for damages. The trial judge nevertheless denied the Alcolac motion in limine for an order to prohibit counsel or witnesses from reference to AIDS as not only irrelevant, but also "notorious and inflammatory." The motion was denied, but the objection saved.

The plaintiffs respond that the experts, including Alcolac witnesses, approved the term chemically induced AIDS to describe the immune suppression conditions of the plaintiffs. Indeed, the question posed by counsel - "[I]s this phenomenon [chemically induced AIDS] supported in the medical and immunological literature?" - evoked from immunologist Zahalsky the response: "It certainly is." The explanation that followed, however, was ellipitical and vague. It did not say that chemically induced AIDS was a medical disease or even a syndrome. It explained only that the National Institute of Environmental Health Sciences, prompted by the need to study the action by toxic chemicals on the immune system, has recently issued the publication "Immune Toxicity" on the subject. Contrary to assertion of the plaintiffs, none of the Alcolac experts acceded that medicine or science acknowledged chemically induced AIDS as a disease, syndrome, or other cognizable phenomenon. They did agree that both viral AIDS and CIID are acquired immune deficiency syndromes marked by gross imbalance between the helper and suppressor populations of the T-cells, but not that the onset or effect of disease are the same.

The response of the plaintiffs reduces, in effect, to the argument that since AIDS and CIID are both acquired immune deficiency syndromes and both are marked by the T-cell imbalance, both are benign synonyms for for the other, so that no prejudice can result from the interchange of use. It was explicit in the testimony of the experts, however, that both terms are not inter-

changeable as a matter of medical definition. Although both expose the victim to opportunistic disease [as does any disregulation of the immune system], AIDS victims display distinctive manifestations, while the effects of CIID are not readily identifiable. Also, AIDS is transmissible, CIID is not. The risk of AIDS is from sexual and drug use practices, the risk of CIID is from exposure to toxic chemicals. AIDS is invariably fatal, CIID is not. See Surgeon General's Report on Acquired Immune Deficiency Syndrome, (U.S. Department of Health and Human Service, 1988).

The sexual and drug use practices usually identified with AIDS remain subjects of public disapproval, even opprobrium. The physical appearance of the victim of the advanced disease can be pathetic. The inevitability of death cannot but engender fear in the victim. A jury cannot but be moved to sympathy for those who, without social blame, have become victim to such a disease, and cannot but be inflamed to punish a defendant whose misconduct induced the havoc of AIDS.

The pervasive use of chemically induced AIDS for chemically induced immune disregulation was unnecessary and gratuitous because the scientific and medical evidence taken most favorably to the verdicts already proved that it was exposure to the Alcolac chemicals that induced the CIID condition in all but two of the plaintiffs. And, as to them, there was competent opinion that the immune system disregulation would manifest later. There was proof, moreover, that Lisa Turley, Lyle Turley and other plaintiffs already manifested those recurrent infections and opportunistic diseases to which victims of suppressed immune systems are vulnerable.

We are left to conclude that the purpose of the AIDS epithet was to inflame the jury to a heavier award of punitive damages than a fair assessment of any found misconduct would

otherwise justify. That purpose shows from the remarks of counsel on closing argument. That effect shows from the request by the jury during deliberations, and granted by the court, for the IMMUNE CHART BLOW UP WITH PEOPLES NAME IN RED WHO MAY HAVE CHEMICALLY INDUCED AIDS - a reference to exhibit 118. The trial court refusal to exclude the term from the proceedings was prejudicial as it impinged on the issue of punitive damages. The awards of punitive damages, as well as of actual damages, were set aside by the trial court, and the appeals of the plaintiffs seek to reinstate them. Accordingly, we defer assessment of the prejudice we find until the determination of those appeals.

c.

Evidentiary Errors

In addition to the claims of evidentiary errors already considered as assertions for new trial, Alcolac argues that the hypotheticals to Dr. Carnow and other experts were faulty and otherwise improperly received. As to Dr. Carnow, Alcolac argues that the hypotheticals were improper in form and content, posed no actual question, and received no answers. In short, they were merely lengthy recapitulations of the favorable evidence without purpose or function. The record discounts that contention.

As to Dr. Bregman and Dr. Reid, Alcolac argues that hypotheticals to them for opinion should not have been allowed because they were not disclosed under Rule 56.01(b)(4)(a) as experts expected as witnesses at the trial. Bregman and Reid were principals with WAPORA, the environmental management subsidiary of Alcolac. Bregman was president of WAPORA and was involved with the design of the Alcolac plant, and so had personal knowledge of the facility from design to actual operation. Reid was an engineer with WAPORA, expert in the treatment of toxic waste. Alcolac called upon WAPORA to investigate the complaints of odors from the

Alcolac operation and to evaluate the malfunctions of the waste disposal plant. They were in fact experts but their testimony was about what they observed, learned and knew about the operation of the plant and the shortcomings of the environmental control system. Alcolac does not say why the Bregman testimony as to the attitude of Alcolac president Anderson was improper. That testimony simply related an attitude unwilling to spend the money necessary to restore the Alcolac environmental control system to proper function - the very purpose Alcolac sought the advice of WAPORA. Alcolac points to no hypothetical question posed to Bregman, nor to any opinion improperly received. The point is without merit.

Alcolac complains also that the trial court erred in the refusal of a mistrial after the testimony of Kenneth Teeters was improperly received. Teeters was a former Division of Health employee and served that agency on 1979. In the course of that duty he investigated a number of complaints about the Alcolac toxic chemicals. He recorded his observations and the narratives of complaints from interviews with area residents, and compiled them as an official report. Those narratives were statements which described the odors, and the annoyance and effects from exposure to them. The persons interviewed included a number of the plaintiffs as well as nonlitigant residents of the environs. The reports compiled by Teeter were qualified under the Business Records Act and received as evidence by the court. Teeter then read to the jury selected portions of the exhibit. Teeter was still a witness when the trial adjourned for the noon recess, and before the proceedings resumed, the court came to the conclusion that numerous excerpts - such as descriptions of the effects of exposure to the chemicals on their persons and on the fauna and flora - were the kind of hearsay the Business Records Act does not sanction as competent evidence. When the trial resumed, the court

by formal instruction withdrew from jury consideration the Teeter testimony of the statements attributed to other people in the report from which he read.

Alcolac argues that the jury had listened to the incompetent testimony for some time, and so the withdrawal instruction did not cure the error. There was, however, virtually nothing the Teeter testimony related that the jury had not heard before or would not hear thereafter. The witnesses the report identifies as the source of the hearsay statements were all presented at the trial and were subject to cross-examination. At least one of them was presented by Alcolac. Many of them related what they had told Teeters. The hearsay evidence at worst was cumulative, and only harmless error. In the circumstances, the withdrawal instruction sufficed as redress and the mistrial was properly denied.

Alcolac also ascribes as error the denial of a motion for mistrial based upon alleged misconduct of the investigator for the plaintiffs. The motion rested on affidavits of Lizzie Barnett, a nonlitigant resident in the environs of Alcolac, and that of her daughter, Lana Davis, who was present at the time of the described incident. It was the tenor of these affidavits that one Ed Bruns, investigator for the plaintiffs, visited Barnett on three occasions to importune her as a witness for the plaintiffs, and on a fourth occasion by another investigator. Bruns handed her a check for $25, a pink piece of paper, and a subpoena. She then asked Bruns, the affidavit recites, "what was in this for [me]," and Bruns then promised $100 additional for her testimony." The affidavit of the daughter confirmed the recital of the mother, and added that the investigator described Mrs. Barnett as "the main witness."

The trial took up the motion before proceeding further with the litigation, but Alcolac rested on the affidavits. The plaintiffs presented one Ramirez, the investigator who had visited

Barnett on the fourth occasion. It became evident through his testimony and explanatory comments that counsel for the plaintiffs had come to Sedalia in preparation for the trial, then at hand, to meet with the numerous persons who were expected to testify as to the Alcolac operations. Lizzie Barnett was among those expected, but she had not appeared. That was the purpose for the visit to the Barnett home of investigator Ramirez. He explained that the $25 tendered to Lizzie Barnett with the subpoena was the witness fee the law requires attend that process. The "pink piece of paper" the Barnett affidavit describes was the standard "instruction to witnesses" form devised by counsel for plaintiffs. It advised that only wages actually lost by the occasion to give court testimony would be reimbursed, that the obligation of the witness was to tell the truth, and to acknowledge forthrightly to any question that the witness had been interviewed about the substance of the expected testimony. Ms. Barnett returned the subpoena, the pink paper and the $25 check to the investigator because she was unable to read them.

It was the Alcolac ground for the mistrial motion that the encounter of the investigators with Ms. Barnett proves intimidation and suggests "that there are other witnesses who may fall in the same category" - so that the integrity of the judicial process was compromised. The intimidation, Alcolac argues, is evident from the comment the Barnett affidavit attributes to investigator Bruns: that Bruns told her "he was going to tell her neighbors that she was not cooperating with plaintiffs" and that "the judge was mad at him because she did not want to testify."

The trial court found no intimidation of any witness or other conduct by the investigators which compromised the judicial process, and overruled the motion for mistrial. The court determined, rather, that any apprehension Ms. Barnett felt from these encounters was not the result of intimidation, but from a mistaken

notion of the nature of the litigation process. The court observed that all of the neighbor witnesses called and to be called were subject to cross-examination, so that Alcolac was free to probe if Alcolac had any intimation that any of them "has been compromised or intimidated into showing up to testify, or had been paid outrageous sums of money to testify in a certain way." Indeed, cross-examination before the hearing on the motion and afterwards explored those subjects, but without effect.

The motion for mistrial was for the trial court to decide as a matter of discretion. The denial of the motion rests on substantial evidence and was not otherwise an abuse of discretion. Hoene v. Associated Dry Goods Corp., 487 S.W.2d 479, 485[11] (Mo. 1972).

8.

Disposition of the Motion for New Trial

The trial court properly denied the Alcolac motion for new trial on all issues of the negligence causes of action.

II

The Nuisance Causes of Action

Twenty-two of the plaintiffs presented evidence to prove that the unreasonable use of its property by Alcolac caused injury to their property.[92] The causes of action were submitted on the theory of private nuisance by MAI 22.06. The jury returned verdicts for the eleven couples in sums which ranged from $3400

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[92]Clarence and Betty Elam; John and Gwen Lawrence; Glen and Bernice Miller; Carl and Ethel Berry; Carl and Jacqueline Berry; Virgil and Dorothy Bradley; Ralph and Genevieve Withers; Dainie and Mary Landon; John and Charlotte Phillips; Arnold and Joyce Sommers; Edward and Malva Gehlken.

[John and Charlotte Phillips] to $15,400 [John and Gwen Lawrence], and aggregated $110,595.

Alcolac argues that the nuisance claims were not submissible and were otherwise tainted by trial error. Alcolac argues more specifically that there was no substantial evidence to prove the unreasonable use paragraph Third of the private nuisance verdict directors ascribes to Alcolac. Instruction No. 24, the prototype verdict director, submitted:

> First, Clarence A. and Betty Elam used their property at 1400 East Boonville as a residence, and
> Second, defendant Alcolac, Inc. operated a chemical plant in close proximity to such property, and
> Third, toxic chemicals escaped from Alcolac,Inc.'s premises onto such property and this substantially impaired plaintiffs' use of their property and damaged plaintiffs' property, and
> Fourth, such use by defendant Alcolac, Inc. of its property was unreasonable.
> [emphasis added]

Alcolac argues that there was no competent proof that toxic chemicals escaped from its premises onto the land of any plaintiff, or injured the use of that property. The argument asserts that, although there was recurrent testimony that noxious odors invaded the property of the plaintiffs, there was no substantial proof that the odors were toxic. We have determined that the evidence abundantly proves those elements of the negligence causes of action and so, coincidentally, of the private nuisance causes of action as well. The private nuisance submissions were for property damage only. Thus, the additional testimony of dwellings made less comfortable, enjoyable and convenient by the invasion of the Alcolac toxicant emissions proved prima facie submissions of private nuisance. Bower v. Hog Builders, Inc., 461 S.W.2d 784, 794 (Mo. 1970).

Alcolac argues nevertheless that the causes of action were not proven because there was no competent evidence that any damages accrued from toxic chemicals during the period used by the appraiser witness to arrive at the opinion of lost value. The causes of action for damage to property were proven and submitted as permanent nuisances. The measure of damages for permanent nuisance is the difference between the fair market value before and after the injury. Spain v. City of Cape Girardeau, 484 S.W.2d 498, 504[4] (Mo. App. 1972). That element of the causes of action was submitted by instructions on the MAI 4.02 prototype.[93]

The opinions of diminished fair market value to the real property of the several plaintiffs from the existence of the permanent Alcolac nuisance were rendered by Teddy Jack Blaylock, real estate broker/appraiser and member of the American Institute of Real Estate Appraisers. His membership connoted competence in the appraisals of all types of real estate. The witness owns and operates Cannon and Blaylock Realty in Columbia, Missouri, a real estate sales and appraisal firm. The firm conducts real estate appraisals throughout the state of Missouri and maintains, as well, an appraisal service - under the supervision of the witness - in Sedalia. His appraisals in the Sedalia area have been sufficiently numerous to enable him to compile records "as to what property had done in the Sedalia area" in past years.

The expert appraiser used the comparable sales or market data method to arrive at the opinions of value as to the several properties. That method measures value by a determination of what

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[93]The litigants assume (evidently as a matter of trial convenience) that the Alcolac operation constitutes a permanent nuisance. They assume also that MAI 4.02 is the proper instruction to measure damages in such a case. But see Scantlin v. City of Pevely, 741 S.W.2d 48, 51[7] n.4 (Mo. App. 1987); Spain v. City of Cape Girardeau, 484 S.W.2d at 505[5]; Stewart v. Marshfield, 431 S.W.2d 819, 822 (Mo. App. 1968).

comparable properties sell for in the marketplace: the appraiser infers the fair market value of the affected property from sales of comparable property. Mo. Condemnation Practice § 5.9 (Mo. Bar 1973, 1979). The difference in the fair market value of comparable property before the event which constitutes the taking and of comparable property after the event determines the loss or increment in the value of the affected property.

Alcolac commenced operations in April of 1978 and the effects on the enjoyment of their property from the toxic emissions were already known to the plaintiffs by July of 1978. As the basis for the opinions of value under the method used, therefore, appraiser Blaylock adopted April of 1978 as the before date and July of 1978 as the after date. Blaylock explained the reasons for the choice of the July date.

> It was chosen merely as the date immediately following commencement of the operation of the Alcolac plant, which would enable me, as the appraiser, to isolate any changes that might be taking place within the neighborhood, except for the introduction of the Alcolac plant.
>
> In other words, there could be no changes in streets, no changes in the character of the properties that were being appraised, no changes in interest rates, or in general economic conditions, or in the economic conditions within Sedalia, and, so, that date immediately following was chosen as the after date.

The witness confined the investigation into comparable sales to an area within four square miles of Alcolac. He resorted to the usual sources for potential comparable sales: information from real estate brokers and appraisers active in the vicinity, his own experience of sales and appraisals of property in the vicinity, the tax assessment rolls of the county, real estate title and tax records, and also the information imparted by the plaintiffs-property owners. That research disclosed that during

the year following the installation of Alcolac, real estate sales were "extremely sluggish" in those four sections [square miles] of land. Blaylock explained:

> [W]ithin that neighborhood, immediately following the introduction of the Alcolac plant, in 1979, the year following, there were only two sales of residential real estate within those four sections of land that could be considered as arm's length transactions, sales between a buyer and seller, not interfamily, but instead, were residential sales.

There were a total of five transfers "of all types of real estate within those four sections of land" in 1979, four in 1980, six in 1981, and two in 1982. They were not all comparable sales.

It was evident to the appraiser that there were not enough comparable sales immediately after July of 1978 to come to a valid opinion of fair market value of the properties affected by the Alcolac presence. In order to arrive at sound after value opinions of the affected properties at all, the witness concluded, it was necessary to consider comparable sales even after several years of July of 1978 in sufficient number to ensure more validity to the opinion of value. That was accomplished by the paired sales technique - the data derived from comparable properties sold twice within the period of research [here, from 1976 through 1985]. The other data for the opinion of values derived from the other, single, comparable sales. In this manner, the value trends of comparable real property were established and a basis for opinions of value of the affected properties in July of 1978.

One value trend was that comparable property sold for more in 1985 than in July of 1978. Another value trend was that the prices of real estate throughout central Missouri in calendar years 1978, 1979, 1980 - and into 1981, increased from 10 to 15 percent per year from inflation. The prices fell some in 1982, but left a net 25% gain. Another value trend was the rates of

interest at an historic high which inhibited sales and at the same time increased the cost to a buyer able to buy. The witness calculated the appreciation in the cost of comparable real estate over the period of research at 65%, but nevertheless attributed the "very conservative" appreciation of only 47% to the properties studied and sales compared to arrive at his opinions of the values of the affected properties in July of 1978. In that manner, all possibility of "false appreciation" was eliminated as a factor of value even as to "the most conservative of buyers." It was by this method of market value trend between July of 1978 and 1985 that the after fair market values of the affected properties were determined and the losses to the plaintiffs assessed. In view of the market value trends during that period - especially the inflated cost of properties from the rampant inflation and historic interest rates - it would be "[b]oth unfair and preposterous," the witness explained, "to subtract a 1984 [or 1985] figure from an April of 1978 figure" to arrive at a before and after market value as a basis to assess the gain or loss to the affected properties. It was the sense of the testimony and opinions of the appraiser that the method used was the most feasible to determine that gain or loss in terms of post-July, 1978 dollars.

The trial court concluded that it was valid for the appraiser, by means of market value trends and comparable sales to explore the full effect of the Alcolac activity as a depressant on the market value of property from July of 1978 [when it first became measurable] until 1984 [when it was fully developed] to derive the after market value of the affected properties - and so allowed the opinions of market value, before and after. The matter of admission of value opinion based upon comparable sales is one of trial court discretion and, under the facts in evidence, falls within the principle of State ex rel. State Highway Comm'n v. Wertz, 478 S.W.2d 670, 675[6,7] (Mo. 1972):

> '" Where evidence of a comparable sale of lease is offered, the trial judge may, in his discretion, admit or exclude it considering such factors as time of the transaction, size, shape and character of the comparable land, and whether there has been any enhancement or depression in value. It makes no difference whether the transaction occurred before or after the date of condemnation so long as it is not too remote a period of time and the land is reasonably comparable, having been neither enhanced or decreased in value by the project or improvement occasioning the taking. The weight to be given such evidence is for the jury. The trial judge's determination as to admissibility or nonadmissibility of such evidence will not be upset on appeal unless it is a clear abuse of discretion.'"

The value opinions of appraiser Blaylock were properly received.

We reject also the insistent and redundant argument Alcolac makes that there was, in any event, no evidence that "any plaintiff's use of property was damaged by toxic chemicals within this 3-month period" - that is between April of 1978 and July of 1978. Our discussion on the negligence causes of action that from the very commencement of the Alcolac operations the plaintiffs were at least discomforted, and already felt physical effects, of the toxic emissions from that source.

The nuisance causes of action were proven and properly submitted.

We reject also the assertions of trial error Alcolac contends prejudiced the property damage verdicts so as to require new trials. Those contentions for new trial were those asserted as to the nuisance causes of action and rejected. The two claims of error our opinion sustains - the exploitation of the evidence of unquantified risk of cancer and of a condition called chemically induced AIDS - impinge only on personal injury compensatory and punitive damages, and not at all on the property damages awards returned on the nuisance causes of action.

The judgments entered on the property damage awards returned on the nuisance causes of action are affirmed.

PART THREE

Appeal of the Plaintiffs

After a trial of more than four months, the jury returned verdicts against Alcolac and awarded to each of the thirty-one plaintiffs on the negligence causes of action for personal injury, $200,000 as compensatory damages and $1,387,096.07 as punitive damages. The court set aside all the awards of both compensatory and punitive damages. The ground for the order as to compensatory damages was that identical verdicts under the evidence most favorable to the plaintiffs were inconsistent with and irreconcilable to each other, and thus impermissible as a matter of law. That ground was also declared to set aside the awards of punitive damages. The punitive damage awards were set aside on additional grounds: 1) the aggregate amount of punitive damages exceeds "by almost three times the net worth of [Alcolac] and thus [were] impermissible as a matter of law" because such verdicts amount to confiscation of Alcolac's property without due process of law and 2) punitive damages amount to penal sanctions, so the submission of that issue by MAI 10.02[94] conjointly with MAI 3.01,[95] which requires only a simple belief by the jury that Alcolac acted

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[94]MAI 10.02 is the punitive damage verdict director in a negligence case. See Notes on Use (1983 Revision).

[95]MAI 3.01 is the general burden of proof instruction in civil cases.

with complete indifference to or conscious disregard for the safety of the plaintiffs rather than by beyond a reasonable doubt,[96] deprived Alcolac of equal protection of the laws.

The memorandum appended to the judgment explained that the order to set aside the awards of actual and punitive damages in the negligence cases and for a new trial on those issues was not an exercise of discretionary power, but rested on legal grounds alone: that the verdicts were "illegal per se."

We affirm the judgment to set aside the awards of actual and punitive damages in the negligence cases and for a new trial limited to those issues, but expressly repudiate that those verdicts as returned by the jury are illegal per se, as the judgment of the trial court has it. We decide rather, that the reception into the proceedings of evidence and argument of unquantified risk of cancer and chemically induced AIDS as elements of personal injury actual and punitive damages was prejudicial to the verdicts, and that the failure of the trial court to grant a new trial on damages on those grounds was error.

I

Identical Verdicts as Ground for New Trial

A

Actual Damages

The memorandum of judgment found the negligence personal injury verdicts, for punitive as well as actual damages, illegal per se because all thirty-one plaintiffs were awarded identical sums. The memorandum reasoned that the evidence was that the plaintiffs were of different genders, ages, life habits, medical histories, life expectancies, exhibited disparate complaints,

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[96]The burden to prove a charge beyond a reasonable doubt is the onus the law imposes upon the prosecution in criminal cases. MAI-CR 3d §§ 300.02 and 302.04.

received disparate diagnoses and prognoses - therefore, the verdicts returned in identical amounts were not only inconsistent with the evidence, but irreconcilable with each other, and so illegal per se. The premise of the order, therefore, is the fact of thirty-one identical verdicts. That is to say: the illegality the court discerns is that, although the evidence of damages was not the same as to all of the plaintiffs, the award of damages was.[97]

In fact, there was a remarkable commonality of disease among the thirty-one plaintiffs attributable by expert opinion to the prolonged exposure to the Alcolac toxins. They all suffered acute effects to the upper respiratory system, or the skin, or to the eyes. The liver organ systems of all of them were affected by disease or dysfunction manifested by chemical hepatitis, or abnormal porphyrin metabolism, or abnormal lipid metabolism, or abnormal protein metabolism - in some combination. The immune systems of all but two of them were either suppressed, disregulated or in dysfunction. The seven males who submitted to the test of the reproductive function all manifested some abnormality. The peripheral nervous systems of a preponderant number of the plaintiffs were diseased from the exposure, as were the 8th cranial nerve - with attendant loss of hearing. These, and other diseases and bodily dysfunctions were not only demonstrated by laboratory tests, but diagnosed as existent, irreversible, and progressive. To be sure, as the trial court order and memorandum of judgment comments, the degree of disease diagnosed as well as the prognosis differed among the plaintiffs, as did the life

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[97]The strict logic of the order would sustain, rather than annul, the identical punitive damages awards since they rest on identical evidence that, as to all the plaintiffs, Alcolac acted with "complete indifference to or conscious disregard for the safety of others" - the jury finding prerequisite to a return of punitive damages under MAI 10.02.

expectancies, and hence the cost of medical care - all elements that bear to prove damages. Even as to one item of reasonably certain future medical need [the surveillance of the damaged immune systems of the plaintiffs], the provable expense, and hence damage, varied according to the life expectancy of each plaintiff. That ranged from 10.67 years for 75-year-old Virgil Bradley to 71.06 years for eleven-year-old Amber Cross.

That said, the order of the trial court to set aside the thirty-one verdicts as "illegal" because the damage awards as to all the plaintiffs were identical, although the evidence was not, remains in error. The rigid logic which fashions the order slights the degree of freedom and discretion our system of law accords a jury on issues of damages.

> It derives first of all from the institutional arrangements under which the jury is permitted to deliberate in private and to report its decision out by general verdict . . . Its basic device is the general damage instruction itself which can do no more than convey to the jury the large headings under which it may award damages. Since these are so broad the chief message of the instructions to the jury is to tell them how free they are.

Kalven, The Jury, The Law, and the Personal Injury Damage Award, 19 Ohio St. L.J. 158, 160 (1958). MAI 4.01, under which the damage awards were assessed by the jury, is of that ilk.

This action was brought as thirteen petitions by [then] thirty-two plaintiffs, each petition a multiple-count suit by members of a family. The petitions were consolidated as a single action under Rule 66.01 by order of the trial court on the motion of Alcolac. Alcolac cannot argue that - had each plaintiff brought a separate petition on which each, in turn, an award of $200,000 as actual damages was returned by the jury on different evidence -the verdicts would have been "inconsistent" and hence

"illegal." Alcolac can no more argue that as to separate suits consolidated for convenient judicial management and tried as one than as to separate suits tried seriatim.

In Page v. Hamilton, 329 S.W.2d 758 (Mo. 1959), our supreme court posed the question, at 765, as

> "whether a result, legally acceptable when achieved by different verdicts in separate trials on the same set of facts, becomes erroneous because of inconsistency when accomplished by a general verdict in a single trial of separate causes of action joined in the same suit by authority of [court rule]. . . .

and responded, at 766[10, 11]:

> "If we adhere to a rule of consistency in this situation the alternatives would be to separate the trials and permit to be done indirectly that which is unseemly if done directly; or to retry the joined causes repeatedly until a verdict is returned which is acceptable to the court's view of consistency.
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> . . . .
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> "'The answer to this argument must be that the law imposes no requirement of consistency upon jurors hearing separate cases which are consolidated for purposes of trial. If such separate cases were being tried separately, by different juries, there would be no assurance of consistency in the verdicts, and no greater assurance of consistency is insisted upon when one jury tries both cases together.
>
> . . . .
>
> "'Inconsistency in verdicts in actions tried together is not a ground for setting aside the judgment in one of the actions on appeal unless such judgment is based on error.'"

See also Brown v. Michigan Miller Mut. Ins. Co., 665 S.W.2d 630, 636 (Mo. App. 1983).

That is to say, the jury is free to report its decision of damages by general verdict on its view of the evidence. And, ordinarily, a general verdict is conclusive as to the questions of

fact properly submitted. In that exercise, no party "has any priority or pre-emptive right to particular evidence or inferences to be drawn therefrom." Page, supra, 329 S.W.2d at 766[10,11]. Nor - as Alcolac suggests - are the private deliberations which culminate as a general verdict open to inquiry or impeachment for faulty logic, misconceived evidence or mistaken calculations. These remain matters which "'"[rest] alone in the juror's breast."'"[98] Elmore v. Owens-Illinois, Inc., 673 S.W.2d 434, 439 (Mo. banc 1984); Anderson v. Independent Mut. Fire Ins. Co., 453 S.W.2d 609, 614[4-7] (Mo. App. 1970). Alcolac cites Claspill v. Craig, 586 S.W.2d 458 (Mo. App. 1979), where the court set aside twelve verdicts of $500 for each of the twelve defendants on their counterclaim. Contrary to the tenor of the Alcolac argument, however, the verdicts in Claspill were annulled not because they were identical, but because the evidence of damage was too "sketchy and vague" to prove $500 as to any of the counterclaimants. 586 S.W.2d at 461[3,4].

> There is no contention here [the claim for increased risk of cancer disallowed] that $200,000 in actual damages was not proven even as to a single plaintiff. Nor can there be. The

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[98]Alcolac argues: "It cannot be doubted, nor is any other conclusion permissible, that the jury did not give the evidence the fair, dispassionate, considered exercise of judgment required." It is not, as we note, an allowable appellate exercise to peer into the privacy of jury deliberations to explore for illogical, mistaken or careless assessment of evidence to explain awards of damages. The overt conduct of the jury nevertheless dispels the contention that the jury was indifferent to the evidence before it. The deliberations extended over three days, and during that course made six separate requests to the court for exhibits, among them: photographs of the plant, the liquid incinerator, WAPORA documents and reports, Alcolac documents and reports, laboratory test results, immunologist Zahalsky's charts, and other such data material to the issues of liability and damages. If such musings were permissible, the uniform awards were more plausibly explained by the Alcolac insistence that all thirty-one claims be submitted and argued together, rather than by separate closing arguments on damages, separate deliberations and, hence, verdicts.

various manifestations of bodily disease and dysfunction diagnosed as to each of the plaintiffs by Dr. Carnow, and reported in the detail of our opinion, rest on substantial evidence.[99] Quite apart from the irreversible bodily damage and disease diagnosed and proven distinctively for each of the plaintiffs, the experts agree that surveillance for early detection of further disease as to persons chronically exposed to chemicals such as the Alcolac toxins is a necessary medical procedure. That surveillance, the experts agree, includes annual panels to monitor the immune system - a bodily function many of the identified Alcolac toxins adversely affect and suppress. That procedure alone, according to immunologist Zahalsky, entails a cost of $1000 per person at the current rate. There were those - Virgil Bradley, Dorothy Bradley, Malva Gehlken and Dainie Landon - who already exhibit severe immune system dysfunction so as to necessitate biannual panels for the rest of their lives. That surveillance alone, over their life expectancies comes to no less than $20,000 for any of the plaintiffs and, for Amber Cross, more than $70,000.[100] This accumulated evidence, no doubt, dissuaded the court from reposing its order for new trial on damages "on the sufficiency of the evidence or the weight thereof."

The freedom and discretion of a jury to return an award of damages under a general verdict, nevertheless, are not beyond some control by the court. It may be set aside as against the weight of the evidence - an option of discretionary action the

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[99]Our delineation of the individual diagnoses does find that a few of the manifestations of disease or dysfunction attributable to Alcolac do not rest on substantial evidence, and notes them. They are not significant to this discussion.

[100]Those plaintiffs with among the lowest life expectancy - Virgil Bradley, 10.67 years, Dorothy Bradley, 13.05 years, Dainie Landon, 11.26 and Malva Gehlken, 20.10 years - were those whose immune systems had already so deteriorated as to require biannual test panels.

trial court explicitly spurned. There were other alternatives.[101] The order to set aside the actual damage verdicts rests, rather, unconditionally on the perceived "illegality" of thirty-one identical awards - notwithstanding the sufficiency of the evidence to sustain them all. The jury awards for actual damages were legal and the order of the trial court to grant new trials on the issue of damages on the ground ascribed was error.

B

Punitive Damages

An award of $1,387,096.07 as punitive damages was returned for each of the thirty-one plaintiffs. The identity, and hence illegality, of the verdicts apart, the court set aside the awards on two legal and expressly nondiscretionary grounds: 1) the assessed punitive damages in the aggregate of $43,000,000 exceeds the Alcolac net worth by almost three times and hence confiscates property without due process of law and 2) punitive damages is a penal sanction found and imposed by a jury upon simple belief, rather than beyond a reasonable doubt, and so deprives a defendant of the equal protection of the laws.

The ground of the order to set aside the punitive damage awards as confiscatory in the aggregate rests on the premise of the memorandum of judgment that the net worth of Alcolac was $15,300,000. There was no evidence before the jury of the Alcolac

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[101] At the time of the order which set aside the damage awards, one of the few traditional means open to a court to control the jury action on damages - the remittitur - had been abolished. See Firestone v. Crown Center Redevelopment Corp., 693 S.W.2d 99, 110 (Mo. banc 1985). The power of a court to order remittur was thereafter restored by § 537.068, RSMo Supp. 1987.

net worth.[102] There were occasional chamber chats on the subject, and the representation of net worth by counsel for Alcolac, but no evidence of the fact of a $15,300,000 net worth. Alcolac argues that the court was not limited by evidence of net worth to act on the jury awards, but that a reference in the record suffices to inform the court for judicial action. Alcolac alludes to two record sources which sufficiently informed the court for the determination that the awards in aggregate so exceeded net worth as to amount to confiscation. One source was the deposition of Alcolac president Anderson where the witness gave the net worth of the defendant, and the other source was the Alcolac, Inc. Annual Reports for 1979 and 1980, marked by plaintiffs as exhibits, but never tendered or received in evidence. We do not know what the Anderson deposition estimate of net worth was,[103] nor was the net worth reported in the Alcolac Annual Reports for 1979 and 1980 extrapolated or explained.

The Anderson deposition was neither in evidence nor part of the record on appeal by any estimate of that term, nor is there any intimation that the trial court was informed of its content. Nor were the Alcolac annual reports in evidence. Alcolac argues that whether in evidence or not, the trial court "did in fact review [the annual reports] on his own," and so had a sufficiently informed basis to annul the punitive damages award. We do not know that the trial judge "did in fact review" the annual reports,

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[102]Evidence of net worth is relevant on the issue of punitive damages, but not prerequisite to an award. Mullen v. Dayringer, 705 S.W.2d 531, 536[11, 12] (Mo. App. 1985).

[103]The deposition of Alcolac president Anderson was not in evidence, no doubt, because he was presented as a witness at the trial. Anderson was also a certified public accountant and shared the responsibility for the Alcolac budget and was qualified to testify as to the net worth of the corporation. Neither the plaintiffs nor the defendant, however, undertook to elicit that evidence from him - for the reasons our opinion makes out.

or if he did, that he came to a determination of net worth from that source, or even what that determination was. There is simply no discernible basis either in the evidence or in the record on appeal for the fact of a $15,300,000 net worth from which the court infers confiscatory punitive damages. There is only the insinuation of that estimate as a fact by counsel for Alcolac.

The want of proof on that issue, however, was not a matter of neglect, but of strategy. The source for that proof [the Alcolac annual reports] and the witness able to interpret those reports [Alcolac president Anderson] were at hand, but the fact of the Alcolac net worth was never elicited. The plaintiffs did not tender the annual reports for proof of net worth because they distrusted the integrity of those financial statements. Alcolac did not tender any evidence of net worth, presumably, to avoid any untoward inferences of hostile examination. In fact, the plaintiffs called Anderson as a witness and examined him on the annual reports, but for another reason. The aim of examination was to establish that his emoluments, in addition to salary, included an incentive bonus based upon 2 percent of the Alcolac consolidated net income. From that, further questions undertook to create the inference that Anderson [an architect of the Alcolac budgets] neglected the WAPORA recommendations for environmental control systems in order to ensure greater incentive bonuses for himself. The questions also undertook to create the inference that the annual report figures were contrived to meliorate the incentive bonus, and so the financial reports lacked integrity. [104]

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[104]The distrust of the integrity of the financial statements these practices engendered, the plaintiffs state in their briefs, prompted them not to offer the annual reports into evidence. They state also that, had Alcolac introduced the reports or other evidence to prove a net worth of $15,300,000 [or, we assume, any comparable worth], the plaintiffs were prepared to show that the Alcolac assets were regularly siphoned by the foreign parent conglomerate, Rio-Tinto Zinc, a corporation with a net worth "measured in billions of dollars." There is nothing in the

There was no evidence of Alcolac net worth before the jury, nor was there any other reliable source for that fact available to the trial court. It is open to a trial court to interfere with a punitive damage award where "it plainly appears that the verdict was so out of all proper proportions as to reveal improper motives and an absence of honest exercise of judgment by the jury in its rendition." Jones v. West Side Buick Auto Co., 231 Mo. App. 187, 93 S.W.2d 1083, 1089[13,14] (1936); Peak v. W.T. Grant Co. 386 S.W.2d 685, 694[20-21] (Mo. App. 1965). The order of the trial court, however couched, rests on the failure of the jury to exercise an honest judgment on evidence it never heard.[105] The premise of the order - that the punitive damage awards in the aggregate exceed the Alcolac net worth by more than three times and so are confiscatory - is without basis in the evidence or record, and so is erroneous.

The order of the trial court sets aside the punitive damage awards on yet the other constitutional ground that the instructions upon which the jury gave redress denied Alcolac the equal protection of the laws - in that the defendant was subjected to punitive sanctions under instructions couched in terms of

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evidence or record on appeal, as such, to validate that assertion. There is a lengthy affidavit of counsel for plaintiffs which appends the reply brief. It details certain "facts" gathered during intensive pretrial discovery: the purchase of Alcolac in 1981 by Rio-Tinto Zinc Corporation, PLC, an English company, the identity of management, the net worth of Rio-Tinto Zinc of Two Billion Three Hundred Fifty-Four Million Four Hundred Thousand Pounds Sterling, the "directing upstream of" dividends from Alcolac to Rio-Tinto, and the "complete authority and control" of Rio-Tinto to approve, modify or reject the Alcolac operating budget. Other than for the acknowledgment at the trial by Anderson that Alcolac was owned by Rio-Tinto, however, these assertions by Affidavit remain only insinuations of counsel, as does the Alcolac representation of a $15,300,000 net worth.

[105] In fact, during the deliberations the jury expressly requested the Alcolac annual reports, but were properly denied by the court: "Dear Jury - The exhibit you have requested was read from only, and not offered or received in evidence. Therefore, I cannot send it to you."

burden of proof applicable to civil tort and not to criminal tort. The punitive damages element was submitted to the jury by verdict director MAI 10.02. The burden of proof was submitted by MAI 3.01. MAI 3.01 instructs that the jury may return a verdict based upon a simple belief of the propositions necessary for verdict. MAI 10.02 is the model for punitive damages based on negligence and allows the return of award "if [the jury] believe" that the conduct of Alcolac, as submitted in the negligence verdict director,

> showed complete indifference to or conscious disregard for the safety of others, then in addition to any damages to which you may find plaintiff entitled under [MAI 4.01] you may award plaintiff an additional amount as punitive damages in such sum as you believe will serve to punish defendant and to deter defendant and others from like conduct. [emphasis added]

The memorandum of judgment not only grounds the equal protection deficiency on the application of the civil burden of proof to the return of a penal sanction, but also on the vagueness of the direction to the jury that the punitive award may be in such sum as the jury believe "will serve to punish defendant and to deter defendant and others from like conduct."

It is evident that this articulation of judgment, whatever its constitutional garb, is an attack on the validity of the model instructions promulgated by our supreme court for the submission of punitive damages in negligence cases. A court of appeals has no authority to declare invalid a model instruction duly promulgated by the supreme court under its power to establish rules of practice, procedure and pleading granted by Article V, § 5, Constitution of Missouri of 1945 as amended August 3, 1976. Any such rule may be annulled or amended only by the supreme court or by a law limited to that purpose.[106] Nor, since such a rule has

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[106]Mo. Const. Art. V § 5 (1945, amended 1976).

the force and effect of a statute,[107] and the supreme court is vested with exclusive appellate jurisdiction in all cases involving the validity of a statute, [108] may we entertain the question the judgment and Alcolac pose.

Our affirmance of the trial court judgment for remand of the negligence causes of action on the issue of damages only, in any event, rests on grounds of testimony prejudicial to the jury treatment of punitive damages, and not because of identical damage awards or constitutional infirmity of instructions. We observe that Alcolac cites no decision of our courts or of the United States Supreme Court[109] which disallows punitive damages in a civil case as a constitutional infringement or even as a matter of unfairness. We notice, rather, that newly enacted § 510.263, RSMo Supp. 1987 assumes the continued efficacy of punitive damages as a redress for aggravated tortious conduct, restores the power of remittitur, and adopts other procedures meant to ensure even more just adjudications of the issue.

C

Submissibility of Punitive Damages

The trial court order does not posit, nor does Alcolac argue, that the plaintiffs failed to show as to the defendant that "complete indifference to or conscious disregard for the safety of others" MAI 10.02 requires a jury to believe as the premise for a punitive damages award. In that context, complete indifference or

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[107]State ex rel. Peabody Coal Co. v. Powell, 574 S.W.2d 423, 426[3,4] (Mo. banc 1978).

[108]Mo. Const. Art. V § 3 (1945, amended 1982); State v. Blackmon, 664 S.W.2d 644, 648[6-8] (Mo. App. 1984).

[109]The question was presented to the United States Supreme Court most recently in Bankers Life & Casualty Co. v. Crenshaw, ___U.S. ___ (1988), 108 S.Ct. 1645, 56 U.S.L.W. 4418, but was refused decision as not properly raised as a federal question.

conscious disregard, our supreme court en banc holds in Hoover's Dairy, Inc. v. Mid-America Dairymen, 700 S.W.2d 426 (Mo. banc 1985), means [at 436[17,18]

> the defendant knew or had reason to know that there was a high degree of probability that the action would result in injury.

We deem that the evidence proved the negligent conduct submitted: that Alcolac loaded the bioponds beyond their design capacity, allowed unfiltered toxic waste to discharge directly into the bioponds, operated the liquid incinerator in such a manner as to discharge toxic chemicals into the atmosphere, and failed to prevent stored chemicals from escape into the atmosphere, and that at the time Alcolac acted [or failed to act] Alcolac knew or had reason to know that there was a high degree of probability that its conduct would result in injury. We deem, therefore, that the issue of liability for punitive damages was submissible on the evidence before the jury and, as our opinion determines, was concluded by the verdicts.

Alcolac commenced production operations at the Sedalia site in May of 1978. Even before construction, at the stage of design, there was great concern over the effect of the production process on the environment. That was because, as Alcolac vice-president and director of operations explained, numerous of the chemicals used in the monomer process were hazardous and toxic. The types of chemicals and raw materials that would be used in production, as Farmer reported to the DNR for licensure before operations commenced, included allyl alcohol, dimethyl sulfate, epichlorohydrin, and those other compounds identified as toxic and even carcinogenic in the later evidence. It was openly acknowledged not only by Farmer, but all of the management cadre, that numerous of the monomer chemicals were hazardous and toxic. In his comments to the townsfolk and officials of Sedalia called

rior to the commencement of operations to allay environmental oncerns,—Farmer explained that "a large percentage of these aterials create either toxic, hazardous, flammable, even carcino- enic conditions." His reassurance to them was, as was his testi- ony, that "these problems" would be managed by the environmental ontrol systems designed for the plant. There can be no doubt, herefore, that Alcolac had actual knowledge that their monomer roduction process used and created toxic and hazardous chemicals hich - if allowed to emit from the premises and expose persons in he environs to them - was attended by a high degree of probabil- ty that injury would result. We accept as foregone, in any event, that a producer who installs an environmental control system to treat toxic wastes already knows the danger to others not involved in the production process from exposure to those wastes.

That is only to say, however, that by the nature of the activity carried on by the defendant, any breach of ordinary care by Alcolac to persons in the environs was attended by a "high degree of probability that the action would result in injury." Those exposed to the Alcolac acrylates, for instance, experienced the injury of skin, eye, nose and respiratory irritations - even if those emissions were only carelessly released. However, "[s]omething more than the mere commission of a tort is always required for punitive damages. There must be circumstances of aggravation . . . . " Prosser and Keeton, The Law of Torts § 2 (5th ed. 1984). In our law, to prove punitive damages, the negli- gent conduct must amount to a "complete indifference to or con- scious disregard for the safety of others." MAI 10.02. It is negligent conduct marked by reckless indifference to the rights of others which proves punitive damages. Hoover's Dairy, Inc. v. Mid-America Dairymen, 700 S.W.2d at 435.

The evidence for punitive damages submissions was abundant:

Alcolac was designed as a state of the art zero discharge plant. Odorous, toxic and hazardous vapors, liquids and sediments were waste products of the soap and monomer processes. It was the theory of the design that any such wastes would be contained within the plant for harmless disposal. Whatever the purpose of the design. Alcolac never operated as a self-contained, closed system. From the first, toxic and odorous wastes were emitted by Alcolac into the atmosphere over the environs in the form of haze, smoke, suds, vapors, acid mist and residues.

There were many reasons.

The intricate, state of the science environmental control system installed in the plant were meant to be operated by an environmental control engineer trained for that function. It was put in the charge rather, first in the hands of a person who was discharged for drunkenness, and then in the charge of a youth - a maintenance employee and high school dropout without any formal training or instruction. It was not until July of 1980 that a trained engineer, Mark Blowers, was given that duty. [He remained for two years]. In the course of the two years between May of 1978, when the Alcolac operations commenced and July of 1980 when Blowers assumed charge of the environmental control building, the vital controls did not function properly as a closed system. The acid scrubbers in the soap building did not function much of the time, so that the vapors were not cleansed before emission into the atmosphere, but remained odorous and caustic. The liquid discharged into the bioponds from the soap factory remained full of suds and became a source of odors as well as a means of transport around the country side of the toxic compounds from the monomer building with which they mixed. The carbon filter beds regularly shifted so that the wastes went through to emission

without adsorption of the toxic contaminants, contrary to the purpose of a closed system. The liquid incinerator, designed as the means of disposal of the waste stream of toxic particulates from the monomer process failed in function in December of 1978 -only seven months after commencement of production - but was continued in use, and belched toxic smoke and haze into the atmosphere.

Alcolac failed as a closed system also because the personnel were neither trained nor encouraged to contain the toxic pollutants within the plant for treatment and disposal. There was no safety program, nor training, nor operation manual to guide them. Toxic chemical spills were common, and were often pushed into the drains with squeegees, rather than through the filtration beds before emission into the biopond. The toxic liquid wastes from the monomer process were also regularly routed directly into the biopond without prior decontamination. From there, they evaporated into the atmosphere, mixed with the suds to be borne around the area, or remained in the biopond to combine with other effluents from the monomer process to synergize into even more potent toxicity. The excess vapors and fumes of sulphur trioxide from the soap plant were merely released into the atmosphere by opening the factory doors. There was no plan to capture and treat the toxic vapors from the raw materials stored on the premises or transferred from transports into the plant. They were stored on the Alcolac grounds in thousands of drums, and not within a structure, so that the toxic fumes and liquids from the frequent leaks from corrosion or mishaps with transport trucks were not contained, but simply evaporated into the ambient air or seeped into the ground.

Alcolac failed as a closed system also because the production consistently exceeded design. The liquid toxic waste generated by production simply was beyond the capacity of the

bioponds to biodegrade. Instead, a sludge of toxic sediment formed and engendered not only odors, but dangerous chemical mixtures and vapors.

These effects were not merely the results of careless lapses in machines, methods and management. They were consequences of deliberate business decisions taken by Alcolac. A jury could have understood from the evidence that the decisions to forgo an environmental control engineer, the redesign of the bioponds to accommodate the increased organic load from the increased production, the correction of the acid scrubbers, the installation of hydrocarbon monitors, and other pollution control measures were prompted by the motives of Alcolac president Anderson to spare expense in order to enhance the profit on which his bonus depended.

There was other evidence from which a jury could find that Alcolac knew that these and other mispractices and deficiencies in the environmental control system were, unless corrected, a continued threat and danger to persons exposed to the chemical wastes which resulted, but that Alcolac, with complete, reckless - even arrogant - indifference, nevertheless allowed them to go uncorrected.

The complaints of the residents around Alcolac of injurious fumes, haze, vapors and odors from the plant began soon after the commencement of production in May of 1978. Those complaints never abated and came to the attention of the Missouri Department of Natural Resources. Odors from the Alcolac premises were palpable, the liquid incinerator generated haze into the atmosphere, the bioponds gave off the scent of hydrogen sulfide and other chemicals, suds blew off the bioponds onto the properties in the environs, and caustic vapors and sediments irritated the skin, eyes an respiration of those on whom they impinged. Citations for excessive emissions issued against Alcolac, as did

notice of violations. As a consequence, the efficacy and operation of the Alcolac environmental control system came under the scrutiny of DNR as early as September of 1978, only four months after the commencement of production. It was then that, for the first time, was mentioned the installation of a recording hydrocarbon detector on the stacks to analyze the organic content of the emissions. There were other elements of the environmental control system that DNR deemed needed further attention.

Alcolac called in WAPORA, its subsidiary - an environmental consultant with international reputation - to investigate and make suggestions. WAPORA reported in the summer of 1978 the problems for correction: No trained competent engineer responsible for the pollution control systems; lack of monitors at the vents to identify and measure any chemicals which might be escaping; the recurrent overload of the bioponds from excessive production as high as eighteen times beyond design capacity; the liquid incinerator malfunction so that toxins escaped into the atmosphere; training of employees and instruction in the Environmental Control Systems Operating Manual devised for the plant by WAPORA to correct the current haphazard handling of chemicals - and other problems. Anderson of Alcolac in the meanwhile asked Bregman of WAPORA to meet with an environmental committee of Sedalia citizens to allay their concerns. Bregman understood that the WAPORA recommendations would be implemented by Alcolac, and so assured the citizens that the air pollution problems would be resolved. The recommendation were rejected, however, by president Anderson as too costly. Anderson then dismissed WAPORA altogether from the Sedalia project in December of 1979, the problems went uncorrected, and the pollution continued. Thus, Anderson not only gave no heed to WAPORA, an affiliate Alcolac management considered its "in-house consulting group," but also discontinued its services.

Anderson did call upon Bregman once again after the commencement of these suits, but to ask that he destroy the WAPORA file on the Sedalia project. Bregman refused.

The pollution from Alcolac did not abate, nor did the effects, and the complaints continued. The environmental engineer WAPORA deemed so essential to the management of the pollution control system was finally employed in mid-1980 and remained for two years. But the employee mispractices and equipment failure continued, and so did the toxic pollution. The liquid incinerator still malfunctioned, the bioponds still gave off odors and suds, the employees still opened the doors to vent spills and excess vapors into the atmosphere rather than to dispose of them within the closed system, and they still routed the monomer wastes around the filtration environmental controls directly into the bioponds where they evaporated or were carried away in the suds. The bioponds were never monitored for toxicity, nor were the vapors or fumes at the points of emission. The liquid incinerator malfunctioned since December of 1978 - only seven months after commencement of production - and continued into year 1981. It was the function of that system to destroy the toxic organic contaminants from the monomer process so that a harmless vapor remained for emission through the stack. The malfunction of the incinerator meant that any matter injected for disposal would only partially burn, or not at all, so that the toxins emitted without change or, if partially changed, into countless toxic components. The malfunction was so complete that Alcolac vice-president Bouroff officially commented to Alcolac president Anderson:

> Our liquid incinerator has the poorest performance ever seen. Most of the time it is out of commission due to all kinds of mechanical problems. It is an absolute waste of time and money. We consider it a 100% failure and the most costly operation. Also, part of our environmental problems were/are caused by this piece of equipment. [emphasis added]

That memorandum was dated May of 1980. Notwithstanding the environmental dangers a malfunctioned incinerator entailed, the use continued for more than a year. In fact, the liquid incinerator had been "out" since December of 1978, as an earlier memorandum from Bouroff to Anderson advised. Thus, for almost three years, instead of harmless vapors, the liquid incinerator emitted into the atmosphere and exposed persons in the environs to a multifarious assortment of toxins and carcinogens. A jury could find from that and other evidence that Alcolac was not only negligent, but acted with complete and reckless indifference to the rights of those around them.

To finally resolve the problem of odor emissions, Alcolac entered into an abatement agreement with the Missouri Department of Natural Resources and undertook nine actions. Among them were the agreement to install a hydrocarbon monitor to sample the emissions from each stack of the vapor incinerator and carbon adsorption system in the monomer building, and to implement a system to determine on a one-time basis the nature and concentration of the emissions from the stacks of the vapor incinerator and carbon adsorption system <u>for each product manufactured in the monomer building</u>. Alcolac also agreed to develop a plan for the training of employees as to the control of odorous emissions from sources at the facility. The abatement agreement was entered on May 30, 1980. The hydrocarbon monitor with alarm was to be installed by January of 1981. It was not fully installed until May of 1984 - more than three years later, and more than six years after the first recommendation for such a pollution control by DNR to Alcolac. The system to determine on a one-time, product-by-product basis the nature and concentration and nature of the emissions from the stacks was <u>never</u> installed. Thus, there was no

way to identify or measure with any definiteness the kind of concentration of any chemical emission from the monomer process. Alcolac did ultimately, as required by the abatement agreement, develop an approved plan for the training of employees as to the control of odorous emissions at the facility. The odorous emissions continued nevertheless, as did the intrusions of foam, the spills at the plant, and the official notices to Alcolac of excessive emissions. These continued almost to the date of trial in 1985.

This evidence, once again, suffices to prove conduct by Alcolac, not merely negligent, but reckless and utterly indifferent to the rights of the plaintiffs. The submissions for punitive damages were proper.

PART FOUR

The Appellate Judgment

We affirm the judgments of the trial court, but for the reasons our opinion gives.

The judgments for property damage entered on the verdicts returned on the nuisance causes of action are affirmed.

The judgments for new trials on the issue of damages only on the negligence causes of action are affirmed.

Our opinion determines, as do the judgments for partial new trials, that the liability of Alcolac to the plaintiffs was proven by substantial evidence and adjudicated without significant error. Our opinion determines, therefore, that as to the negligence causes of action, Alcolac breached its duty of care to the plaintiffs, and that such neglect caused them personal injury. In terms of a toxic tort negligence cause of action, this means that all the components of cause in fact have been proven and adjudicated without significant error as to each plaintiff:

> [1] exposure by the plaintiff to an identified harmful substance[s] significant enough to

activate disease.

[2] a demonstrable relationship between the substance[s] and biological disease

[3] diagnosis of such disease in the plaintiff

[4] expert opinion that the disease found in the plaintiff is consistent with exposure to the harmful substance[s]

[5] defendant was responsible for the etiologic agent of the disease diagnosed in the plaintiff

Our opinion concludes, as do necessarily the judgments for partial new trials, that the diagnosis of biological injury, disease and dysfunction Dr. Carnow entered for each of the thirty-one plaintiffs as attributable to exposure to the Alcolac toxins is derived by valid scientific principle, based on competent scientific proof, and rests on substantial evidence.

What remains upon remand is to determine the money damages due each plaintiff as compensation for the personal injury already proven, and the amount of punitive damages. The manifestations of disease and dysfunction from the chronic systemic chemical intoxication diagnosed by Dr. Carnow as the biological damage to each plaintiff caused by the Alcolac toxic chemicals and entered on the DIAGNOSIS chart [except as we presently note] rest on substantial evidence and define the bodily injury proven. We find three lapses in the otherwise meticulous proof of the manifestations of biological damage attributable to Alcolac: There is no medical basis for the manifestation of reproductive system dysfunction entered for Dainie Landon. He could not produce a specimen for fertility analysis, and hence that manifestation does not rest on substantial evidence and was not proven. There is no medical basis, by test result, clinical procedure or other explanation for the abnormal protein or abnormal prophyrin metabolism components of the diagnosis entered for John Lawrence. Those

entries were mistaken and were not proven. There is no medical basis of explanation for the hypertension - mild entered for Bernice Miller, and hence that manifestation was not proven.

To facilitate the task of the jury upon an eventual retrial, and to subserve the efficient judicial management of this still massive litigation, the court may set aside, suspend, or modify the order of consolidation of the thirty-one claims and submit the issue of damages by the single claim, or other judicial unit, as convenience and fairness may prompt. Wolfner v. Miller, 711 S.W.2d 580, 582[1,2] (Mo. App. 1986); Keeling v. Randall, 386 S.W.2d 67, 69[4] (Mo. banc 1965); Rule 66.02. The provisions of § 510.163, RSMo Supp. 1987, also bear on the trial procedures where punitive damages are an issue for adjudication. In such cases, a party may request the bifurcation of the issues of liability for and award of compensatory damages from the issues of liability for and award of punitive damages for separate trial. The liability for compensatory and punitive damages is already concluded. What remains of this litigation is for the jury to adjudicate the amount of compensatory damages and of punitive damages due each plaintiff. To subserve sound judicial management and principles of fairness, the court may - even absent request -dissever those issues for separate trial and submission to the jury. State ex rel. Blond v. Stubbs, 485 S.W.2d 153, 158[10] (Mo. App. 1972); Rule 66.02.

It is a desideratum of the statute [§ 510.263.4] that a defendant not be required to pay redundant punitive damages based on the same conduct. To accommodate that end - in these circumstances of a liability for punitive damages already adjudged in favor of thirty-one plaintiffs - the court may aptly under Rule 66.01(b) submit to the jury for verdict the common issue of the

sum that will serve to punish and deter Alcolac and others from like conduct [MAI 10.02] rendered as a single sum and apportioned separately by verdict as to each plaintiff.[110]

The costs of the action are assessed two-thirds against the defendant Alcolac and one-third against the plaintiffs.

Charles Shangler
Presiding Judge

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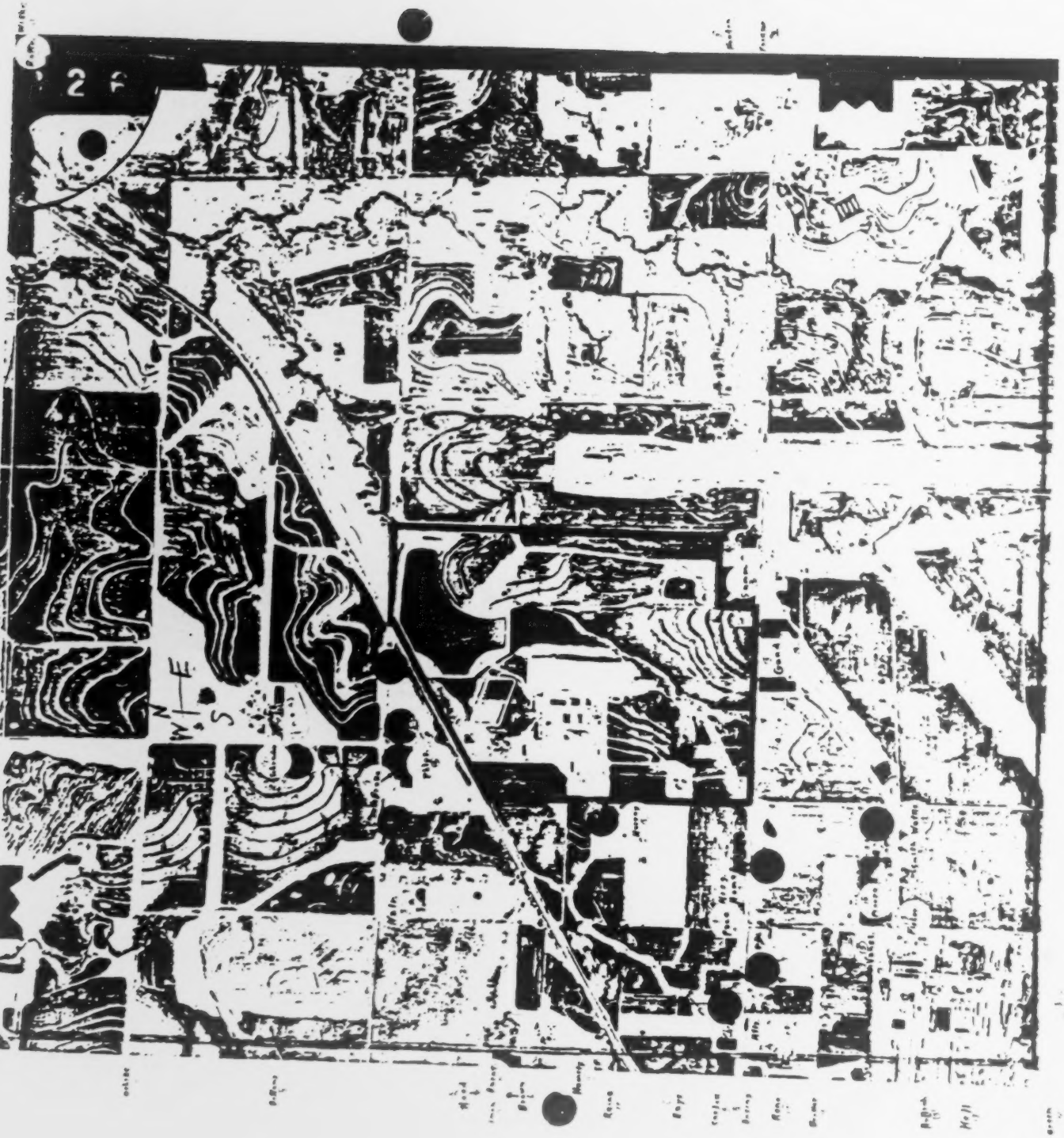
[110]In circumstances such as here of multiple claims or multiple parties, under Rule 74.01(b) a jury verdict as to part of a claim or some of the parties does not terminate the action or issue as a judgment. In the absence of a specific determination by the court to the contrary: "[A]ny order or other form of decision, however designated, that adjudicates fewer than all the claims or the rights and liabilities of fewer than all the parties shall not terminate the action as to any of the claims or parties . . . ." The practical consequence is that, contrary to the effect of former rule and procedure, the motion for new trial and other post judgment events do not commence to mature until the action is terminated as to all the claims and all the parties. See Nanette K. Laughrey, Judgments--The New Missouri Rule, 44 Mo. B.J. 11, 12-13 (1988).

APPENDIX A




PLAINTIFF'S EXHIBIT

APPENDIX B



APPENDIX C

APPENDIX E

Berry family group

Carl D. Berry and wife Ethel [age 73] lived on their 3 1/2 acre tract since 1942. The land lies some 3/4ths of a mile south of Alcolac. Carl was disabled from some cause extraneous to the suit and joined only for remedy for damage to the real estate. He died during the pendency of appeal and wife Ethel succeeded to that interest. Carl C. Berry [age 54] and wife Jacqueline [age 55] live on the tract which adjoins the parental land on the northeast.

Ethel Berry

She has lived on the property since 1942 and has never detected any odors from Missouri Pacific Railroad yards, already there. It was not until Alcolac commenced operations in May of 1978 that she was subjected to any odors or fumes or other noticeable emission. Since then until the present odors of rotten eggs, sulphur, and gas had come from Alcolac recurrently, depending upon the wind direction or calm. There have been and continue to be incidents of smoke and mist emissions from Alcolac in the early morning and late at night. A powder covers the ground and vegetation, suds and foam fill the air and alight on her property, and black soot descends on her property from Alcolac explosions.

Inhalation and contact with these emissions cause her eyes to itch and water, sore throats, burning in the nose and chest, nosebleeds, headaches, depression, chills, rashes and blisters over the body, swelling of the legs, numbness, cramps and tingling of the legs, and difficulty in breathing.

She kept a garden, fruit trees, and raised chickens. In April of 1983 after an incident of unusual smoke and fumes, 26 of their 100 chickens died. They also lost some hive bees.

Her prior history includes a condition of arthritis and borderline diabetes.

She consumes no alcohol, nor smokes, and drinks no more than two cups of coffee per day.

She complained to the Sedalia mayor about the odors and fumes, but to no avail.

Carl C. Berry

He and wife Jacqueline have lived on the lot since 1959, but left in 1977 and returned in 1979 and live there still.

When he returned in 1979, the odors from Alcolac were palpable and resembled the scent of lacquer and at times that of a very strong acid. There

was also dark black smoke from the Alcolac operations - fifty or sixty times - which has come over the property and given off a sour odor. A white powder covers the grass and vegetation and black specks cover the car and outer surface of the residence. Foam and suds from the Alcolac ponds rise to a great height, like a "white mountain," travel through the air onto his property in "huge pieces three feet across." There were no such incidents before his return to the property in 1979. The white substance causes a rash on the skin and eyes to burn. The odors cause regular headaches, chest pains, eyes to burn and water, sore nose, rapid heart beat, cramps in arms and legs, skin itch, shortness of breath.
The cattle he keeps are affected by the odors and fumes; all have runny eyes and a sore on one of them does not heal. He no longer keeps a garden or grows crops because the powdery white matter and black specks he attributes to Alcolac impair their growth. Carl Berry is a construction worker who lays asphalt on the highway. He worked for a while as a spray painter, and for a while installed foam insulation. He no longer smokes although he smoked a pack of cigarettes a day for seventeen years, until 1969.
His prior history includes back pain caused by a short leg; an episode of chest pain in 1971, and another from the inhalation of carbon monoxide at work in 1981. He complained to the Sedalia mayor about the Alcolac odors and fumes, as well as to the DNR, but to no avail.

Jacqueline Berry
She noticed the odors and fumes from Alcolac upon their return in 1979. She had never experienced odors during her prior residence there from 1958 to 1977 from any source. She could not describe the odors, other than as obnoxious. They were constant when the wind was out of the north or northeast. At times the haze and vapors from Alcolac were so dense that even the image of the plant was obscured. An explosion at the plant in 1981 blew the roof off Alcolac. She observed "great big bunches" of foam and suds fly by the window and "slam" into their property. The odors were terrible and continued to the time of the trial. The odors caused headaches, eyes and nose to burn, blurred vision, congestion in the lungs and throat, fatigue, shortness of breath, skin rashes, cramps and numbness in the calves, arms and hands, loss of memory and hearing decrease.
She has part-time employment with J.C. Penney. She has smoked half a pack a day for twenty-five years; drinks coffee, and has a drink of alcohol a couple of times a year.

Her prior history includes a lens implant after the removal of a cataract; varicose veins and sinus difficulty.
She reported the odors to Alcolac, the Sedalia mayor and councilmen, but to no avail.

Bradley family group

Virgil Bradley [age 75] and Dorothy Bradley [age 74] lived on the 16 acre tract 4/10ths of a mile north and west of Alcolac from 1955 to 1984, when they moved residence into the city of Sedalia to escape the odors and fumes from Alcolac.

Virgil Bradley
He was employed at the Missouri Pacific Railroad yards in Sedalia from 1947 to 1962. He had never experienced any foul odors from that or other sources until Alcolac commenced operations in May of 1978. He described the odors from Alcolac as the scent of rotten eggs and separately, as a sweet odor. Also, suds and foam from Alcolac floated over and onto their land many times, fragments as "big as a washtub." The odors and fumes cause the eyes to burn, headaches, coughs, sleepiness and dizziness, tiredness, pains in the chest, rashes on the skin, vomiting, tingling and numbness in the hands, legs and feet, pains in the chest and shortness of breath.
The odors are most frequent and intense when the wind is from the south.
He and his wife would escape from the odors by trips away from the home, some times all day long.
The discomfort and annoyance were so great that they moved into Sedalia proper at the end of 1984, but still own the residence near Alcolac. They return there every night to feed the cat, and the odors persist.
His prior history includes the loss of the right arm in an industrial accident and varicose veins.
He does not smoke, consumes no alcohol and drinks a very occasional cup of coffee.
He complained of the odors and suds to the county officials, the DNR, the sheriff and police, but to no avail.

Dorothy Bradley
She first noticed odors from any source after Alcolac commenced operations in May of 1978. The odors were of rotten eggs and of chemicals. There was also haze and smoke from Alcolac, as well as foam and suds. The emissions cause headaches, aches in the eyeballs, vomiting,

sleepiness, sore throat, hoarseness, and loss of memory. The odors are more frequent and noticeable when the wind is from the south. To escape the odors, the Bradleys went to Liberty Park west of Sedalia, and then moved away at the end of 1984. They return daily to feed the cats, but the odors remain.
Her prior history includes a lump removed from the breast.

Elam family group

Clarence Elam [age 64] and Betty Elam [age 59] live on a nine acre tract one-half mile southwest of Alcolac. They have occupied the premises since 1972 with the daughter, plaintiff Linda Lou Elam [now Sanders, age 23], who married, and now lives in Texas.

Clarence Elam
The Elams live about 1/4 mile from the Missouri Pacific Railroad yards and near Missouri Pressed Metals and have never detected any foul odors or scents from those sources. The odors commenced in 1978 after Alcolac commenced operations. The odors were of lacquer and of "hot radiators." There were also episodes of foam and suds which alighted on their property, in fragments as big as a "large refrigerator." There was also smoke and haze from Alcolac. He determined the source was Alcolac alone. The odors resulted in eye irritations, matting of the eyes, chest pains, severe burnings in the throat and nose, drowsiness, lack of energy, impaired memory, dizziness, and watery eyes.
In years 1978 and 1979, he was employed in the installation of missile sites, and so was exposed to the emissions only on weekends.
He kept horses and prize coon hounds on the property. The odors caused the horses to have runny eyes and noses and the hounds to roll, and rub their heads and nose and eyes.
The hounds lost the ability to track.
He was a structural iron worker, now retired.
He worked for a brief period in the demolition of two furnaces which contained asbestos.
His prior history includes a loss of hearing, and a regimen of diabetes medication.
He no longer smokes, although he indulged that habit until he was 40 years of age, uses no alcohol, and consumes three to four cups of coffee per day.

Betty Elam

The odors and fumes come only from Alcolac, and not from any of the other enterprises. She has detected odors and seen suds from Alcolac regularly since May of 1978 to the present. Her experiences with odors, fumes, smoke and haze have been in the hundreds. They include episodes of night burnings she has observed at Alcolac. Very often at night to escape the odors, and while her husband was away at work, she drove to a parking lot or went to Smithton to be with her son.

There were different odors, some were pungent, some sweet, some overwhelming. The odors caused difficulty in breathing, dizziness, blurred vision; numbness and tingling in the hands and legs, itching, nosebleeds, loss of memory, racing heartbeat, nausea, loss of energy, eye irritation, skin blisters and headaches.

She kept a diary of these occurrences, as did most of the other plaintiffs, and marked significant incidents of explosion, unusual vapor mist formations on the property from Alcolac and odors of unusual intensity. They included episodes of foam and suds "as big as a Volkswagen" on her property as well as on the property of others and on the public roads.

Her prior history includes a chronic anal stricture, chronic obstructive pulmonary disease and low back strain. Betty Elam has smoked a pack of cigarettes a day since she was 16. She drinks pots of coffee during the day on medical advice because, she explained, her "body lacks acid."

She has complained repeatedly about the emissions from Alcolac to State Senator Mathewson, to the local environmental control office, to the mayors of Sedalia, to the EPA and to the DNR. Nikkila of the DNR gave her his personal number and encouraged her to telephone if anyone reported an odor from the Alcolac site. Over the past five years, she has called him about severe odors hundreds of times.

Linda Elam Sanders

She resided with the parents until 1980, when she married and moved to San Antonio, Texas. Thus, she lived near Alcolac for a year and nine months. The odors commenced after Alcolac commenced operations and occurred all the time and sometimes every other week. The odors were of rotten eggs and an overbearing lacquer scent. The emissions from Alcolac also included haze and smoke, sometimes very black, a white film on the grass and foam flying in the air.

She has seen the foam on the bioponds. Her complaints are: burning in the eyes, nose and throat, headaches, difficult breathing, coughing, bloodshot eyes, sharp pain under the ear and that her legs go to sleep. Her history includes a heart murmur diagnosed by Sedalia physician Alcorn in 1982.
Linda Sanders smoked occasionally while in high school, but not thereafter. She drinks no coffee and only occasionally, alcohol.

Gehlken family group

Edward Gehlken [age 68] and wife Malva [age 65] took occupancy of their 2 1/2 acre tract 1/2 mile north of Alcolac in July of 1978. The discomfort from the Alcolac odors and fumes prompted their move into the city of Sedalia in April of 1984. Edward became incapacitated from a series of strokes. He was unable to present himself for medical examinations and did not give testimony, and his claim for personal injuries was never submitted. The court entered a directed verdict against his claim, and that judgment, unappealed, is now final.

Malva Gehlken
The Alcolac operations emitted terrible odors and generated suds which came on her property. On one occasion she saw chunks of foam "as big as five gallon buckets" floating through the air and alighting on bushes and trees. On the occasion of the September 25, 1981 explosion at Alcolac, soot and a black flaky substance was blown onto their property. The fumes and odors caused her eyes to water and to become irritated, her nasal passages to become swollen, the throat dry and burned, brought on persistent headaches, shortness of breath, loss of hearing, cramps in the legs and right foot, charley horses in the calves as well as numbness and tingling, change in the pigmentation of the skin and blurred vision.
Her fruit trees and bushes and other vegetation were affected and her garden would no longer produce, although her husband, who tended them, was an experienced farmer.
Her prior history included the anomaly of being born with three kidneys, the tingling and numbness of the wrist since age thirty, headaches in 1970 from a whiplash, and an earlier temporary loss of hearing from medication. She neither drinks alcohol nor smokes and consumes coffee only two or three times a week.

She complained to the DNR about the odors, fumes and discomfort, as well as to the local newspaper, but to no avail.

Landon family group

Dainie Landon [age 74] and wife Mary Landon [age 71] lived on a tract immediately west and directly across the road from the entrance to Alcolac. They moved into a mobile home in May of 1984, prompted by the odors, fumes and other discomforts caused by the Alcolac operation, but retained the residence. Mary Landon had a family history of cancer. She was diagnosed as afflicted with leukemia in the early 1940's, experienced episodes of lymphoma, and in February of 1984 cancer of the colon was found and surgically excised.

Dainie Landon

He commenced to notice odors and fumes in the summer of 1978. Some of them smelled like oily acid. They also saw haze "like a big black rolling cloud" from Alcolac which came in the direction of their property, usually at night. The odors were worst when the winds were out of the north. The Landons often left the residence to escape the odors and smells.

The emissions from Alcolac caused his eyes to water and burn, the throat to burn, and brought on headaches. His nose bled, he felt sickness of the stomach, passed out, the ears stopped up, the lips and face were covered with an oily film, and large rashes and blisters formed on the exposed parts of his body. He felt shortness of breath. He kept a garden, chickens, dogs and a horse. The fruit trees never matured, the tomatoes never matured, the garden was covered with a white film, the horse foundered, and chickens died.

He returns twice a day to feed and water the animals, and the continued odors cause his symptoms to flare up.

His prior history includes a diagnosis in 1973 of emphysema. He worked in iron construction for 34 years and used red lead, a substance which was then prohibited. He smoked a pack of cigarettes per day for 30 years until the 1973 diagnosis of emphysema and drinks no coffee or alcohol.

The Landons have complained to the DNR about the odors, and to State Senator Mathewson, but to no avail. DNR installed a monitor on the premises with instructions to activate the switch when they smelled odors, but the results were never received from Nikilla at DNR as promised.

Mary Landon

The odors commenced in 1978 and gave off the scent of rotten eggs and sulphur. She commenced to record the incidents of odors and emissions from Alcolac on August 8, 1979 and maintained it until May 8, 1984, when they moved into the mobile home. The odors and smoke caused sores on her body, dizziness, drowsiness, headaches, stomach upsets, chest aches, coughs with blood in the phlegm, difficult breathing and swallowing, a fast heart beat, irritated nose and blurred vision.

There were foam storms from Alcolac which came onto her property and which caused swelling and blisters when touched.

Her prior history included a congenital condition of red blood cell anomaly she had mistakenly believed to be a state of pre-leukemia, and for that reason submitted to the removal of her spleen. She was diagnosed for cancer of the bowel in February of 1984 and had surgery and was then treated by cobalt. She lacks a gall bladder, spleen, suffered from thyroid and heart conditions and obesity.

She neither smoked, nor drank alcohol, nor consumed coffee.

She often left the home with her husband to escape the odors and fumes.

She complained of the fumes and odors to State Senator Mathewson, Congressman Skelton, the office of the Sheriff and to Alcolac. Mark Blowers, then Alcolac environmental engineer, invited her to call him if needed. Mary Landon was in communication with Nikkila a number of times, but these and other complaints were of no avail.

She died in May of 1986 while appeal pended.

Lawrence family group

John Lawrence [age 62] and Gwendolyn Lawrence [age 64] lived on a 24 acre tract one-half mile southwest of Alcolac. They acquired the land in 1968 and continue to own the premises. In order to escape the odors and other Alcolac emissions, they moved in with a nearby daughter and returned in 1981. They remained until November 12, 1983, when they left again for the same reasons, and now live six miles west of Sedalia. They continue to visit the home near Alcolac daily.

John Lawrence

The commencement of the Alcolac productions caused odors, smoke, fumes and foam to drift on the Lawrence property. He described the odors

as of rotten eggs, sweet and "skunky." These brought on headaches, blurred vision, watery eyes, vomiting, sickness in the stomach, drowsiness, sleepiness, cramps in the arms and legs, rashes on the skin, tingling and numbness in the arms and legs, itching, matted eyes, burned throat, difficult breathing and deep coughs. These symptoms abated away from the premises but revived when he returned to the property from time to time and inhaled odors and other emissions.
His prior history includes a condition of ulcers, since cured.
He has not smoked since 1977, drinks no alcohol, and consumes three to four cups of coffee per day. He is employed as a maintenance worker at a Sedalia school.
He complained to government officials, the DNR and also to Alcolac president Anderson and plant manager Fischer, both of whom visited the Lawrences at the home and acknowledged the "odor problems," but said that they would be corrected. The odors and other emissions persisted.

Gwendolyn Lawrence
Although the Lawrence residence was located near the Missouri Pacific Railroad yards and other plants, there was no odor from those sources. Odors, smoke, haze, mist and other emissions recurred over the Lawrence property day and night only after Alcolac commenced operations. These emissions were observed to come from Alcolac. The odors caused her eyes to burn, the vision to blur, hoarseness and burning in the throat, nosebleeds, rashes on the skin, leg cramps, numbness and tingling in the arms and legs; stiffness in the joints, headaches, ulcerated nose; pains in the chest, heart flutters, lethargy, itching, weakness in the legs and back leg muscles, tremors in the hand and a lack of muscular coordination. Other emissions from Alcolac manifested as greasy oil droplets on the flowers and other surfaces. There were also foam storms from the bioponds of "big chunks" of suds flying by the picture window and alighting on the property with foam "as high as eight feet" "like balloons in the sky." The emissions continue.
The odors are palpable when the wind is from the north or is calm.
Her prior history includes a goiter condition, an ulcer condition in 1965, and a gallbladder surgery in 1978. In 1982 she was treated in

the hospital for constipation, vomiting and a burning of the esophagus - the latter of which she reported was caused by Alcolac.
She neither smokes nor drinks alcohol, and consumes three or four cups of coffee per day.
She and others complained to State Senator Mathewson about the Alcolac odors, who gave her a credit card to call the DNR about odors. On one occasion in 1978, when her eyes "had been burned real bad," she telephoned Alcolac president Anderson and plant manager Fischer, who came to her home. She was asked to "bear with them" and that they would get the odors stopped. They persisted nevertheless.
She attended numerous environmental quality control meetings of the public, complained to Senator Eagleton, and when all else failed, commenced suit against Alcolac.

Miller family group

Glenn Miller [age 54] and Berniece Miller [age 54] lived on a five acre tract one-half mile north of Alcolac from 1975 until August of 1984, when the recurrence of the odors and physical discomforts they caused prompted them to move to Springfield, Missouri.

Glenn Miller
He kept an organic garden on the property and used no pesticides. The garden was productive until 1978, when spots, suds and other emissions from Alcolac affected the plants. The peach trees produced nodules instead of fruit and the plum tree shriveled and died.
Odors became palpable after Alcolac began operations and smelled of rotten eggs, ammonia and compressed gas. They were especially noticeable when the wind was southerly. There were explosions at Alcolac when black smoke, accompanied by odors, came over his land. The odors sapped his strength, caused the eyes to burn and water, the nose to bleed, numbness in the right leg, nausea, swollen eyes and throat soreness.
His prior history includes varicose veins.
He complained to Nikkila at the DNR that there was a very serious odor problem. Teter of the Division of Health consulted with him about the health effects and the blight on the crops from the Alcolac emissions, but nothing changed. He complained also to Sgt. Rice - then an agent of the DNR - about the odors, but they continued.
He and his wife eventually moved to Springfield, Missouri to escape the effects of the emissions.

Bernice Miller

She never felt any discomfort from any of the business enterprises in the vicinity until Alcolac commenced operations in May of 1978. She began to smell foul odors of rotten eggs, and of hair solution used in beauty parlors for permanents. The intensity of the odors depended upon the direction of the wind and they penetrated into the residence interior. Odors as well as suds came over onto her property. The suds were blown from the Alcolac bioponds, usually about twice a month. There were explosions which dispersed soot onto the property, and smoke and haze drifted over from the Alcolac stack. When the odors became intense, she and her husband left for the home of a daughter or simply took a drive. The emissions caused her eye, throat, and nose trouble, sickening headaches, loss of energy, eye burns and infections, loss of sleep, stiff neck, numbness in the arms, tingling in the hands, impairment of hearing, irritated throat and hoarseness, stiffness and aches in the joints, shoulders and arms.

Her prior history included high blood pressure and obesity. She complained to the DNR and Nikkila and to city officials about the emissions and odors, but to no avail.

Phillips family group

John Phillips [age 58] and Charlotte Phillips [age 52] purchased residence property - an acre and-a-half - in 1966 and continue to occupy the premises. The tract lies one-half mile directly north of Alcolac, separated only by a track of railroad.

John Phillips

Life on the acreage was pleasant until Alcolac commenced operations in 1978. There were never any odors from the Missouri Pacific Railroad yards or any other plants in the vicinity until Alcolac started production. He could not describe the odors, but they are persistent and continue to the present time. They are most noticeable when the wind is from the south or is calm. In addition to odors, a black substance settles on cars and other surfaces, especially after explosions in the monomer plant. Suds and foam from Alcolac alight everywhere and on his property and into the waterway which feeds Shaver Creek. A white substance covers the vegetation and an oily substance covers the garden plants.

The wild life is no longer worth eating. The garden, once productive, and the domestic animals, once edible, have been so affected by the emissions that he has cut down the one and butchered the other.
The odors and emissions have brought on blurred vision, nausea, dizziness, eyes which burn and water, vomiting, numbness and loss of grip in both hands, numbness and tingling in the feet to the knees, numbness and tingling in the right arm, pains in the chest, sore throat, loss of appetite, depression and loss of libido, headaches, pain in the left eye, shortness of breath, loss of hearing and fatigue.
He is a retired construction worker. He smoked cigarettes for forty years until 1981, and now occasionally smokes a pipe. He drinks no alcohol, but consumes six to seven cups of coffee per day.
His prior medical history includes an appendicitis in 1947; an inner ear infection in about 1977; gastritis in 1952 and breathing problems - diagnosed as bronchial obstructive disease in 1981.
He complained to president Anderson at Alcolac about the odors and emissions and to then plant manager Aid, but to no avail. Teeter, with the Missouri Division of Health, advised him to cut down his garden,and Phillips complied.

Charlotte Philliips
She was subjected to neither odors, suds nor other emissions from any source before Alcolac commenced operations in 1978. The odors were of rotten eggs, varnish, "real sickening and sweet" - also, of dead fish and sulphur. Foam and suds came onto her property and near her property "really a lot of times." A black substance or specks fell over the surfaces, most noticeably after the explosion of September 25, 1981. There were also incursions of black smoke from Alcolac. A white substance covered the grass, trees and flowers. These emissions and substances were never present before the Alcolac operations.
She complained of burning and bloodshot eyes, dizziness, nausea, hoarseness, earaches, headaches, fatigue, vomiting, hearing loss, skin rashes and growths, cramps in the calves and legs, sore and burning throat, urinary tract infection and loss of libido.
A horse they kept lost skin off the nose from eating the grass. They cut down their garden on the advise of the Missouri Department of Health and no longer have that source, nor the domestic animals, as a source for food.

She weighs 200 pounds. She is employed as a forklift operator. She does not smoke nor consume alcohol. She drinks two cups of coffee per week.
Her prior history includes a gallstone surgery, a kidney infection, borderline diabetes and a constipation problem related to the colon.
She complained of the odors and emissions to the Mayor and councilmen of Sedalia, to State Senator Mathewson, to Congressman Skelton, Senator Eagleton and to the municipal police and fire departments. They were to no avail. She complained to Alcolac as well,and president Anderson responded by a visit to the house and said he would "try to fix" the odors. The respective plant managers, Fischer and Aid, came a number of times, and both apologized and promised to try a correction, but that never came about. On one early occasion Dr. Boies from WAPORA responded to a complaint of odors and conducted her around the plant to determine the source - it was from a leak in the monomer building.

Pryor family group

Daniel Pryor [age 35] and Joyce Pryor [age 33] lived on premises a mile and-a-half east of Alcolac from October of 1978 to February of 1984, when the marriage was dissolved and they separated. Amber Cross [age 11], a child of Joyce Pryor by a former marriage, was a member of their household. Thus, Amber shared the premises between her fourth and ninth birthdays.

Daniel Pryor
He described the odors generated by Alcolac as that of ammonia and cat urine. There were many occasions of suds that could be seen "all across the road where they were stacked up pretty high." At night as he drove by Alcolac, the atmosphere was always hazy. He developed symptoms never present before Alcolac: dizziness, severe headaches, nausea, fatigue, stomach cramps, dryness of the throat and mouth, stinging, red and running eyes, loss of memory, stiffness in the legs, calves, arm sleeps and tingle and shortness of breath. He works as a carpenter and now smokes half-a-pack of cigarettes per day, drinks three cups of decaffeinated coffee per day and consumes a twelve pack of beer over the weekend and two drinks of hard liquor per day. He smoked marijuana briefly after the Vietnamese war and

experimented with LSD once. He had consumed no alcohol for nine years before 1984, but resumed following the marital discord.
His prior history includes an ear infection and skin rashes during service in Vietnam.

Joyce Pryor
The odors from Alcolac were apparent to her both at home and enroute and return from work by the plant. The suds and foam did not drift as far as their residence, but were to be found on the road nearby, at least once, to a depth of the car bumper. The odors were "real pungent," like ammonia and rotten boiled eggs. She began to experience physical complaints after October of 1978: nausea, diarrhea, rashes, eyes burning, inflamed with a "swollen and ached feeling," dryness of the mouth and throat, a "chemical breath," numbness and tingling in the arm and leg, headaches, impaired hearing, a disrupted menstrual cycle, vomiting, chest pains, disorientation and a rapid pulse. Her personal habits have been spasmodic. She commenced to smoke at age 19, then discontinued from 1975 to 1980 and now uses less than a pack a day. She experimented with heroin and marijuana during her first marriage [to Cross], and was depressed during her marital discord with Pryor. She drinks tea and two liquors a day. Her employment at Missouri Pressed Metals in Sedalia [from October 1978 to June 1979] involved powdered graphite, copper and other metals.
Her prior history includes a nervous breakdown in 1973, and swelling to her hands and face and rashes from the work duty at Missouri Pressed Metals Company. The occupational symptoms subsided after she left that employment, but the physical conditions induced by the Alcolac emissions persist.

Amber Cross
She recalls the foam and suds on one occasion and describes the odors as of burnt tires. She was exposed to the odors when she rode her bicycle on Griessen Road - where her home was located and which bounded Alcolac on the north. She observed foam on the creeks and odors emanated from them as well. When she played in the yard, her nose burned constantly, her head became feverish, and she coughed up phlegm. When she played near the creek, she got headaches, dizziness,and a sense that she was about to faint. She experienced "real bad" sore throats and coughed up food and blood. Her eyes watered and became red and burned; she itched

and rashes appeared on her chest, legs and back. On the occasion of a visit to her step-mother Cross, she gave the child marijuana to smoke and beer and wine to sip.
Her prior history includes a kidney infection, which the physician diagnosed as the consumption of "too much pop."

Sommers family group

Arnold Sommers [age 43] and Joyce Sommers [age 38] live with their daughter Joy [age 15] and his daughter Tammy [age 19] on a 13 acre tract purchased in 1973 and located about 3/4 of a mile southwest of Alcolac. Missouri Pressed Metals is located about 200 feet from the home, and the Missouri Pacific Railroad yard and other plants are close by, but none has been the source of any troublesome odor or emission. It was not until after Alcolac commenced operations that odors and other emollient became palpable and induced physical complaints.

Arnold Sommers
He described two odor-types as most frequent: that of sulphur and of a sweet perfume. There are also emissions of ash or smoke he observes drift down from Alcolac and settle on the garden and vegetation. Suds come over onto the property "like a blizzard." A mist or smoke from Alcolac, so dense as to obscure sight, envelops the pasture. These emissions cause headaches, nosebleeds, numbness in the hands and foot, cramps, charley horses and occasional tingling, nausea, eyes to water and to become matted shut, the throat to burn, sinus trouble, impediment in speech, soreness of the neck and rapid heart beat. The effect of exposure to these conditions caused him a rage so that he struck his horse in the nose with such force so as to break his hand.
The owned acreage was also used to grow food and to raise livestock. The garden no longer produces. Twelve of the thirty pigs died since the onset of Alcolac operations, as well as nine calves and two cows. They have been afflicted with a condition of red, watery eyes and the cattle as well as a showhorse are afflicted with an irritation which causes them to abrade their hides.
He drives a truck for employment, drinks about two beers a day, and has smoked a pack-and-a-half of cigarettes a day since age seventeen.
His past history includes an ulcer condition, a fractured right leg and wrist from a motorcycle accident, a broken nose from another vehicular

accident, and metal has twice penetrated his eye. There have been no permanent consequences from those conditions and misadventures.

Joyce Sommers

She was free of significant physical problems before the advent of Alcolac in May of 1978. Then the incidence of smoke, fumes and odors began. There were also numerous explosions at the Alcolac plant, as well as drifts of foam on their property from that source. The odors were predominantly of sulphur. The exposures from Alcolac caused her nosebleeds, headaches, sinus drain, sore throat, dizziness, loss of stamina, leg cramps with tingling and numbness, recurrence of an irregular menstrual cycle, earaches and irritated skin. The emissions affected the garden and domestic animals also. The growth of the fruits and vegetables was stunted, and the horse and cattle developed sores from constant rubbing.

The last explosion at Alcolac occurred only a few days before her trial testimony - on September 6, 1985 - and was marked by a strong sulphur odor.

She is presently employed by the Sedalia Democrat, the local newspaper and, earlier, worked at the Missouri Pressed Metals, without medical consequences. She missed work in May of 1983 for dizziness caused by a condition of the inner ear.

She neither smokes, nor drinks alcoholic beverages, and consumes coffee only occasionally. Her prior history includes an infection of the urinary tract at age 18 and a broken jaw, dislocated hip and tracheotomy from an automobile accident in 1971.

Tammy Sommers

She left the family residence in Sedalia in January of 1983, more than four years after Alcolac commenced operations. The quality of air up to then was normal, but thereafter, "horrible odors, smoke, foam and soap bubbles" were carried onto the property by the wind. The odors and emissions caused coughing practically daily. The odors and fumes would interrupt her sleep with headaches. She has developed tingling and numbness in the hand and a loss of grip. She has nosebleeds and congestion in the head and nose and recently she has developed pain in the colon.

She is now a mother. She smokes a pack of cigarettes per day and drinks two cups of coffee a day. Her prior history includes a cut left arm and right hand from an encounter with

a plate glass door, and a stitched elbow from a like, later encounter. In 1984 she fractured an ankle and leg in an automobile accident.

Joy Sommers

She was eight years of age when Alcolac commenced production. That is when the odors, fumes, smoke and suds commenced. Her newspaper delivery course took her around the Alcolac plant and the odors from the plant and the foam from the ponds were palpable. The foam often came onto the family property, as did the smoke, and white specks fell on the vegetation. She described the odors as "like burning plastic, sulphur, dead fish, super glue, alcohol perfume." The pond in the pasture, a swimming hole, was covered with a film of oil. The emissions after the onset of the Alcolac operations brought on nosebleeds, headaches, nose congestion, sore throat, earaches, sinus activity and blisters on the body. There were no smoke or odors from the Missouri Pacific Railroad yards nearby or from Missouri Pressed Metals or from the other plants. There is an occasional odor of lacquer from a fiberglass plant nearby, but nothing else.
She has no significant prior history.
The odors persist and were experienced, most recently, the night before she testified at the trial. They continue to awaken her frequently and cause a congestion which makes breathing difficult.

Turley family group

James Turley [age 42] and Kay Turley [age 44] moved onto a 60 acre tract located somewhat more than a mile due west of Alcolac and lived there with the three children, William Lance Turley [age 22], Lisa Turley [age 21] and Lyle Turley [age 9]. Lyle is the child of the marriage and William and Lisa are children of Kay Turley through a former marriage, and adopted by James Turley. The acreage was purchased as a place of retirement upon the conclusion of military service by James Turley, then a lieutenant colonel. The persistent course of emissions from Alcolac and the physical consequences to the members of the family prompted them to move to Abilene, Texas at the end of May of 1981. They sold the residence and a three acre tract on which it rested and still retain 57 acres.

James Turley
His sense of smell was impaired by a series of breaks to his nose, consequently he was unable to detect or describe any odors except those from closeby sources - as from dairy farm operations. He did observe soap foam blow across his land - especially when the wind was from the east - and his personal investigation disclosed that Alcolac was the source. In August of 1978, he commenced to have difficulty in breathing through the nose, and the irritation of burning and constant drainage through the nose became constant. He became lethargic, lacked endurance, his knee became tender to the touch, rashes developed on the skin, as well as tremors from fatigue. His hearing and memory became impaired, as was his libido. A prostate infection developed as well as a continual burning and watering of the eyes.
He neither smokes nor drinks and consumes three to four cups of coffee per day.
His prior history includes a restriction in breathing caused by a series of nose fractures incurred during football scrimmages in his youth which impaired his sense of smell; a spastic colon and the incidence and removal of a polyp in that organ.

Kay Turley
The onset of Alcolac operations brought on numerous physical complaints which included fatigue, congestion of the nose, watering, itching and redness of the eyes, lethargy, pressure in the right ear and depression.
She does not smoke or consume alcoholic beverages and drinks two cups of coffee or tea daily.
Her past history includes a condition of anemia which she treats by normal supplements; an episode of mitral valve prolapse which remains uneventful unless exposed to infection.

William Lance Turley
He was 15 years of age when Alcolac commenced production in May of 1978. He - in contrast to his father who repeatedly suffered breaks to his nose - has a keen sense of smell. The odors from Alcolac were sweet. He complains of tiredness, lethargy and the inability to concentrate, conditions which have impaired his school work. His nose runs and he has colds which persist. The eyes burn, swell and water and his nose bleeds persistently. His throat is sore, the chest rattles with phlegm, he

coughs up blood and mucus, and he experiences numbness and tingling from the elbows to the fingertips and from the knees to the feet. He neither smokes nor drinks coffee, but since his marriage he drinks alcoholic beverages occasionally. His prior history includes a medical opinion in 1971 that he becomes infected easily,and has experienced skin rashes before the advent of Alcolac. He also had frequent ear infections when younger.

Lisa Turley
She recalled experiences of odors - she was then 12 years of age - and described them as rotten odors, but could not then say whether they were related to the Alcolac activity. She complains of pain in the knee joints, feet, arms and hips and aches so severe as to incapacitate her. She is lethargic, lacks energy and falls asleep uncontrollably. She suffers from headaches, congestion, cough and watery eyes.
She does not smoke, drinks alcohol about three times a week and drinks coffee occasionally. Her prior history includes broken arms from a fall from a tree in 1979, and shoulder problems related to her swimming activity.

Lyle Turley
He was five years of age when the family moved from Sedalia to Abilene. He recalls that he coughed a great deal, and that his throat was sore and congested. His mother described his symptoms as that of a "very sick boy": he coughed all night long as though he was trying to dislodge something from his throat, and squinted his eyes a great deal. The father described his symptoms as a persistent hacking cough and a runny nose. These symptoms as well as the squinting of the eyes have been relieved by the move away from the Sedalia property.

Withers family group
Ralph Withers [age 54] and Genevieve Withers [age 54] live on an 8 and 1/2 acre tract just 1/2 mile north and across the tracks from Alcolac. There were no discernible odors from 1959, when they assumed occupancy of the premises, until May of 1978, when Alcolac commenced production.

Ralph Withers
The odors engendered by the Alcolac explosions, smoke and other emissions were those of ammonia, burnt styrofoam and old rotten cabbage. There was an explosion at Alcolac in September

of 1981 when the odors were particularly intense and noxious. There were suds and foam from the bioponds in "gobs as big as a five-gallon bucket" which alighted on their property. Smoke was generated at Alcolac at night and came onto the property. He complained of headaches, stiffness of the shoulders and neck, a throat sore and scratchy, skin rash and a sinus condition aggravated.
He discontinued his pack-a-day cigarette habit in 1967, drinks three to four cups of coffee per day, and imbibes an occasional alcoholic drink.
His prior history includes the removal of a polyp from his throat in 1967, a sinus difficulty, and complaints of chest pain in 1977.
To county official Hieronymous and to Teeter of the Missouri Division of Health he complained about the odors which he described as odors of paint or lacquer. The complaints did not avail.

Genevieve Withers
She described a white substance residue and black specks on the grapes and other herbiage. Suds and foam have come on the property "like a snowstorm" from Alcolac, and explosions have vented black soot, over the garage, patio and car. The last explosions and chemical fire at Alcolac occurred just a few days before her testimony at the trial. Foul odors and smoke also are recurrent incidents of that operation. The odors are of ammonia, rotten eggs, lacquer and rotten cabbage. They cause headaches, aggravate the sinus, cause a sore throat, a reduced stamina, watering and itching of the eyes, charley horses and stiffness in the legs, numbness and tingling in the legs and bladder infections, loss of concentration and memory and diminished hearing. She neither smokes, nor consumes alcohol, and drinks one cup of coffee per day.
Her prior history includes a condition of diabetes, and a condition of high blood pressure.
She complained of the odors and emissions to county official Hieronymous and to Nikkila at the DNR, but to no effect.

From: P. Bouroft

APPENDIX F

May 27, 1980 PROGRESS AS OF MAY 1980

Item 1: 2M1M: One of the "incineratables", methanol 70%, MEK 30% azeotrope once accumulated to a T/V quantity is shipped to Rohm & Haas at no cost to us but freight. This is so far the cheapest disposal proposition.

Items 1 & 2: Our liquid incinerator has the poorest performance ever seen. Most of the time it is out of commission due to all kinds of mechanical problems. It is an absolute waste of time and money. We consider it a 100% failure and the most costly operation. Also, part of our environmental problems were/are caused by this piece of equipment. We suggest buying/installing a new unit, capacity at least double of the present "theoretical" 50 gph.

On June 10-11, 1980, Mr. Larry Doucet, P.E., a world expert in incineration will be visiting Sedalia. He will lecture, evaluate our incinerator (it does not have to be:useless) and recommend the right one. Until then, no action will be taken except to try to operate what we have.

Items 3 & 6: At a cost of 33¢/gallon, dispose of as many drums of wastes as possible by transferring same into hauler's T/V's. This to comply with the DNR request and improve the appearance of our plant. Problem: we have a hard time finding a cooperage firm to pick up the used drums. We are working on it, also H. Caldwell, who was asked to investigate.

As of mid May 1980, we have shipped seven loads of wastes and emptied over 1,000 drums.

Item 3:

a. AGE brine treatment to remove tin: So far about 10 x 3,000 gallon batches in R-600 have been processed as reported by W. Sutton. It is not known how many batches will be required in the 6,000 gallon "tin" tank to treat the contents of two lagoons containing this brine.

Attention W. Sutton: Would you please estimate the number of batches and time needed in the 6,000 gallon tank/reactor.

Presently the "freshly" made AGE brine is treated to partially precipitate tin while steam stripping AA in R-600. This saves time and energy, combining two operations into one. Unfortunately, the brine stored in the lagoons will have to be reheated in the 6,000 gallon reactor.

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C00000000303

DEPOSITION EXHIBIT

at 10 a/s/84

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APPENDIX G

# MEMORANDUM

To: All Employees

From: William Sutton

Subject: Odor and Pollution Control

Date: May 22, 1980

Ref:

Cc:

During the past three months we have been cited three times by the Air Pollution Enforcement Division of the Department of Natural Resources for excessive odor emissions. These citations are serious and will probably result in further action from the DNR. In addition there have been numerous complaints and unfavorable publicity by the local news media.

In depth review of the causes of the citations and complaints reveal that human error in one way or another was involved : Most of these errors were operational and could have been prevented had proper procedures been followed or additional thought given to actions before proceeding. It is not necessary to go into details because I am sure each of you know the incidents that were involved and why they happened.

We are presently in a position that further operating errors or other causes of odors will be very serious. They could result in additional citations and ultimately the shut down of the plant. This of course means our jobs are possibly on the line and points out the real concern that should be applied to the control of odors and wastes.

In practice the control of emissions to the environment is everybody's responsibility and should not be left to the other person. Everyone should be conerned about this problem and are expected to show sound judgement in their actions. Continued errors that cause environmental problems will nave to result in disciplinary action to the offenders.



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DEPOSITION EXHIBIT
11/9/84

APPENDIX H

Odor and Pollution Control
(continued)

I am sure everyone would like to do their part in controlling this difficult problem. Should anyone note a problem with odors or any type of emission, please try to correct the situation or at least bring to the immediate attention of the person in charge of the area for their action. We are open to suggestions from anyone who has ideas that would improve our environmental program.

Your cooperation in handling this serious situation will be greatly appreciated

Bill Sutton

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WHS/djp

ADAPTED FROM USCL
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SHELL OIL COMPANY
SHELL CHEMICAL COMPANY
SHELL DEVELOPMENT COMPANY
SHELL PIPE LINE CORPORATION

MSDS 5080-6

HAZARD RATING

NFPA

# MATERIAL SAFETY DATA SHEET

Information on this form is furnished solely for the purpose of compliance with the Occupational Safety and Health Act of 1970 and shall not be used for any other purpose. Use or dissemination of all or any part of this information for any other purpose may result in a violation of law or statute pounds for legislation.

## SECTION I

| | |
|---|---|
| MANUFACTURER'S NAME: Shell Chemical Company | EMERGENCY TELEPHONE NO.: (713) 473-9461 |
| ADDRESS (Number, Street, City, State, and ZIP Code): One Shell Plaza, P. O. Box 2463, Houston, Texas 77001 | |
| CHEMICAL NAME AND SYNONYMS: Epichlorohydrin, 1-chloro-2,3-epoxy propane | TRADE NAME AND SYNONYMS: Epichlorohydrin, ECH |
| CHEMICAL FAMILY: Chlorinated epoxide | FORMULA: $C_3H_5OCl$ |

## SECTION II HAZARDOUS INGREDIENTS*

| COMPOSITION | % | SPECIES | $LD_{50}$ Oral | $LD_{50}$ Dermal | $LC_{50}$ Concentration | $LC_{50}$ Hours |
|---|---|---|---|---|---|---|
| Epichlorohydrin | 100 | Rat | 90 mg/kg | | 500 ppm | 4 |
| | | Rabbit | | 1500 mg/kg | | |

The liquid can be fatal if swallowed. High vapor concentrations can cause death.

ECH has been reported to produce cancer in laboratory animals and to produce mutagenic changes in bacteria and cultured human cells; however, the results of a recent epidemiology study have been termed "highly suggestive" that exposure to ECH is a cancer risk to humans. ECH is listed as a cancer suspect agent by ACGIH.

## SECTION III PHYSICAL DATA

| | | | |
|---|---|---|---|
| BOILING POINT (°F): | 239 | SPECIFIC GRAVITY ($H_2O$ = 1): | 1.18 |
| VAPOR PRESSURE (mm Hg @ 70°F): | 13.8 | PERCENT VOLATILE BY VOLUME (%): | |
| VAPOR DENSITY (Air = 1): | 3.2 | EVAPORATION RATE (Butyl Acetate = 1): | 1.0* |
| SOLUBILITY IN WATER @ 68°F: | 6.6 | | |

APPEARANCE AND ODOR: Colorless, mobile liquid with an irritating chloroform like odor. Odor threshold is much low. This greatly exceeds the PEL, therefore air monitoring is required to ensure safe levels.

## SECTION IV FIRE AND EXPLOSION HAZARD DATA

| | | | |
|---|---|---|---|
| FLASH POINT (Method Used): 87°F TCC | | FLAMMABLE LIMITS: | LEL: 3.8 UEL: 21.0 |

EXTINGUISHING MEDIA: Alcohol foam, dry chemical, $CO_2$, water spray.

SPECIAL FIRE FIGHTING PROCEDURES: Notify fire dept. Keep containers cool with water spray. Vapors are toxic. Wear protective equipment including NIOSH approved self-contained breathing apparatus. Do not use solid stream of water as it will scatter and spread fire.

UNUSUAL FIRE AND EXPLOSION HAZARDS: A flammable liquid. Vapors form explosive mixture when heated. May polymerize and burst containers. Heating produces HCl. Combustion may also produce toxic and lethal carbon monoxide and phosgene.

*Reported by Shell Oil Company

APPENDIX J

## SECTION V HEALTH HAZARD DATA

THRESHOLD LIMIT VALUE 5 ppm or 19 mg/M³ TWA (OSHA); Shell internal action levels - 1 ppm TWA; 3 ppm short term excursion.

EFFECTS OF OVEREXPOSURE Vapors severely irritating to the eyes, nose and throat. Repeated or prolonged exposure can cause severe and lasting lung, liver and kidney damage and changes in lymphocytes (cells in blood and lymph). High vapor concentrations can be fatal. Liquid can cause severe burns and permanent injury from contact with skin or eyes. Liquid can be fatal if swallowed. (See Section II). Cancer suspect agent (Group 2).

EMERGENCY AND FIRST AID PROCEDURES Remove contaminated clothes immediately. Flush eyes with water at least 15 minutes. Wash skin thoroughly. Get medical attention. If swallowed, induce vomiting except for unconscious victim. Get medical attention. Contaminated leather articles (including shoes) cannot be decontaminated and must be destroyed.

## SECTION VI REACTIVITY DATA

| | | | |
|---|---|---|---|
| STABILITY | UNSTABLE | | CONDITIONS TO AVOID Open flames. Temperatures above |
| | STABLE | X | 600°F, contact with materials noted below. |

INCOMPATIBILITY (Materials to avoid) Acids; Bases; Amines; $NH_3$; Na, Zn, Al, Mg & their alloys; Fe, Al, & Zn Halides

HAZARDOUS DECOMPOSITION PRODUCTS Releases hydrogen chloride, phosgene and CO

| | | | |
|---|---|---|---|
| HAZARDOUS POLYMERIZATION | MAY OCCUR | X | CONDITIONS TO AVOID Contact with incompatible materials shown |
| | WILL NOT OCCUR | | above. |

Penetrates leather easily. Attacks some kinds of plastics, rubber and coatings.

## SECTION VII SPILL OR LEAK PROCEDURES

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED Remove all ignition sources. Evacuate personnel not equipped with protective clothing & NIOSH approved respiratory protection. Absorb small quantities on inert material. Dike and pump or vacuum large quantities into containers. Close securely and label waste containers properly. Prevent entry into SEWERS, CLOSED WATER SUPPLIES AND SEWERS. Notify authorities if this happens or is threatened. The U.S. Coast Guard and EPA emergency number is (800) 424-8802.

WASTE DISPOSAL METHOD Use controlled burning under approved EPA conditions in equipment capable of handling hydrogen chloride fumes.

## SECTION VIII SPECIAL PROTECTION INFORMATION

RESPIRATORY PROTECTION (Specify type) NIOSH approved supplied air respiratory equipment as required.

| | | |
|---|---|---|
| VENTILATION | LOCAL EXHAUST Required for personnel protection | SPECIAL |
| | MECHANICAL (General) | OTHER |

PROTECTIVE GLOVES Impervious gloves | EYE PROTECTION Splash proof goggles

OTHER PROTECTIVE EQUIPMENT Impervious equipment as required to prevent all body contact.

## SECTION IX SPECIAL PRECAUTIONS

SPECIAL RULES TO BE FOLLOWED IN HANDLING AND STORING Store in tightly closed containers in cooled, well-ventilated area. Handled as flammable liquid and as Class B poison. Prevent exposure when transferring material. Wash before eating, smoking or using toilet facilities.

OTHER PRECAUTIONS Notify authorities if any exposure to the general public or environment is threatened by a spill. HANDLE AS A FLAMMABLE TOXIC LIQUID.

Shell Oil Company
Product Safety & Compliance
Oil & Chemical Products
DATE August, 1975

THE INFORMATION CONTAINED HEREIN IS BASED ON DATA CONSIDERED ACCURATE. HOWEVER, NO WARRANTY IS EXPRESSED OR IMPLIED REGARDING THE ACCURACY OF THESE DATA OR THE RESULTS TO BE OBTAINED FROM THE USE THEREOF. SHELL OIL COMPANY SHALL NOT BE RESPONSIBLE FOR ANY INJURY, LOSS OR DAMAGE OF ANY KIND RESULTING FROM THE USE OF THE MATERIAL IF REASONABLE SAFETY PROCEDURES ARE NOT ADVISED TO BE IMPLEMENTED IN THE DATA SHEET. SHELL OIL COMPANY ASSUMES NO RESPONSIBILITY FOR INJURY TO VENDEE OR TO OTHER PERSONS OR PROPERTY CAUSED BY ABNORMAL USE OF THE MATERIAL EVEN IF REASONABLE SAFETY PROCEDURES ARE FOLLOWED. FURTHER, SHELL OIL COMPANY ASSUMES THE RISK IN THE USE OF THE MATERIAL WHEN VENDEE HAS BEEN ADVISED TO SUBSTITUTE IT FOR ANY PURPOSE.

APPENDIX K

Carl Berry

He noted that some of his laboratory tests were "slightly outside the normal range" but could not say "what that means clinically at this point in time." He noted also the Midwest/-Wheeler result of 19.7 for the HNK test with a reference range of 3.8 to 9.6, and deemed it "of some interest." He acknowledged that the "clinical significance of that at the moment is really not clear," but concluded nevertheless: "[H]e doesn't seem to have a clinical immune disease."

Ethel Berry

He acknowledged that "[s]he has some tests that again fall outside the normal range," but did not know "what the significance of those are." He concluded that she had no functional abnormality in her immune system.

Jacquelyn Berry

He noted "a few cell populations that are again outside the normal." He did not think that to be "clinically significant," and found no functional abnormality in her immune system.

Dorothy Bradley

He found an abnormal helper subpopulation [T/4] to a suppressor subpopulation [T/8] ratio - a ratio of 4.03 against a reference range of 1.10 to 2.55. He commented: "[T]he clinical significance of that is highly speculative." He found no functional abnormality as to her immune system. Dorothy Bradley was a plaintiff excluded from the KU protocol, hence the evaluation rests exclusively upon the other tests and medical sources.

Virgil Bradley

He noted "some subtle changes in the lymphocyte numbers." The test results marked a T/4 to T/8 cell ratio outside the reference range. He found no functional abnormality in Virgil's immune system.

Amber Cross

She was among the plaintiffs excluded from the KU tests protocol. The other laboratory tests indicated an abnormal T/4 to T/8 cell population - a value of .95 with a reference range of 1.10 to 2.55. That plaintiff was then eleven years of age, but that abnormality gave the evaluator no concern. The evaluator gave opinion that there was no functional abnormality in the immune system of Amber Cross. He acknowledged that he did not regularly treat children with immunologic diseases.

Betty Elam

He found nothing of clinical significance from the records before, and found no functional abnormality in her immune system.

Clarence Elam

He noted a value for HNK [Human Natural Killer cells] of 12.5, outside the reference range, 3.8 to 9.6, but found it of no significance. He found no functional abnormality in Clarence's immune system.

Malva Gehlken

She was among the plaintiffs excluded from the KU tests protocol. The evaluator found a value below the reference range in the T/4 to T/8 cell ratio. That gave him no cause for concern "in terms of her functional response," and he determined there was no functional abnormality in her immune system.

Dainie Landon

The evaluator found "some abnormal numbers," a T/4 to T/8 cell ratio "slightly above the reference range" - a 2.78 value beyond the outside reference range of 2.55. That was no cause for concern - and presumably his immune system was intact in terms of function.

Mary Landon

The evaluator found that her laboratory tests were "clearly abnormal," "far and away different than what you would expect for an individual her age, sex, etc." He explained that radiation is known to be "particularly toxic or detrimental to the immune system," but could point to no "direct evidence that [her] radiation treatment

has caused the abnormality in her immune system." He concluded, nevertheless, that the abnormalities in her immune system disclosed by the tests were "most likely" the result of the radiation therapy.

Gwendolyn Lawrence

He found nothing on which to comment in her various tests and concluded that she displayed no functional abnormality in her immune system.

John Lawrence

He found an HNK value of 20 with a reference range of 3.8 to 9.6, but determined that although it was "of interest" as "outside what is usually found in people," that finding was of no "clinical relevance." He found no functional abnormality in John's immune system.

Bernice Miller

He found an HNK value of 15 with a reference range of 3.8 to 9.6, but determined it was not significant, and concluded there was no functional abnormality in her immune system.

Glen Miller

He found some tests outside the normal range in the lymphocyte subpopulations, but found no abnormality in the function of Glen's immune system.

Charlotte Phillips

He found nothing in the records to indicate "a significant immune dysfunction or irregularity," and found no functional abnormality in her immune system.

John Phillips

He was among the plaintiffs excluded from the KU tests protocol. The evaluator found nothing in the records to indicate a significant immune dysfunction or irregularity. He found no functional abnormality in John's immune system.

Daniel Pryor

He was among the plaintiffs excluded from the KU tests protocol. The evaluator found from the records some laboratory tests outside the normal range, but found no functional abnormality in Daniel's immune system.

Joyce Pryor

She was among those plaintiffs excluded from the KU tests protocol. He found nothing significant in the records of the other laboratory tests and found no functional abnormality in her immune system.

Linda Elam Sanders

She was among those plaintiffs excluded from the KU tests protocol. The records of the other tests disclosed a slightly depressed immoglobulin level, a matter he deemed without clinical significance. He found no functional abnormality in her immune system.

Arnold Sommers

He was among those plaintiffs excluded from the KU tests protocol. His records disclosed the HNK subpopulation of lymphocytes "slightly outside the normal range." The evaluator did not know the significance of that finding. He determined there was no functional abnormality in Arnold's immune system.

Jov Sommers

He found nothing of significance in her test results, and determined that there was no functional abnormality in her immune system.

Tammy Sommers

He found some of her lymphocyte subpopulations to be outside the normal range, but did not know the significance of that finding. He determined there was no functional abnormality in her immune system.

James Turley

He found no significant immune dysfunction or irregularity from the test records and concluded that James had no functional abnormality in his immune system.

Kay Turley

She was among those plaintiffs excluded from the KU tests protocol. Her records disclosed a history of seasonal rhinitis - an inflammation of the nose - in this case, an allergic reaction. He found no functional abnormality in her immune system.

Lyle Turley

He was among the plaintiffs excluded from the KU tests protocol. He also had a history of seasonal, allergic rhinitis. The evaluator found no functional abnormality in his immune system.

Lance Turley

He found no significant immune dysfunction or irregularity from the records and concluded Lance had no functional abnormality in his immune system.

Genevieve Withers

He noted an HNK test result value of 3.3 with a reference range of 3.8 to 9.6, but found, nevertheless, no functional abnormality in her immune system.

Ralph Withers

He found no immune dysfunction or irregularity from the test records. The evaluator determined that there was no functional abnormality in Ralph's immune system.

# DOROTHY BRADLEY

AGE 74
LIFE EXPECTANCY 13.05

SYMPTOMS

E
WATERING
BLURRED VISION
RED, IRRITATED, ETC.

R
DISCOMFORT OR PAIN
TINNITIS
●DECREASED ACUITY

SE
NOSE BLEEDS
NASAL CONGESTION
IRRITATED NOSE
SNEEZING

ROAT
●SORENESS OR DIFFICULTY SWALLOWING

LMONARY
SHORTNESS OF BREATH
● COUGH
WHEEZE
●CHEST HEAVINESS
HEMOPTYSIS

DIOVASCULAR
PERIPHERAL VASCULAR
CHEST PAIN
ARRHYTHMIA

STROINTESTINAL
● GASTROINTESTINAL DISTRESS
● VOMITING
● NAUSEA
HEMORRHOIDS AND RECTAL BLEEDING
CONSTIPATION
DIARRHEA
ANOREXIA
WEIGHT LOSS

ITOURINARY
POOR CONTROL
PAIN OR DIFFICULTY IN URINATION
FREQUENCY
NOCTURIA

**REPRODUCTIVE OR SEXUAL DYSFUNCTION**
DECREASED LIBIDO
IMPOTENCE, MALE
MENSTRUAL DISORDERS, FEMALE

**CENTRAL NERVOUS SYSTEM**
DIZZINESS OR LOSS OF CONSCIOUSNESS
●DECREASED CONCENTRATION OR MEMORY LOSS
DEPRESSION
● NERVOUSNESS
SLEEP DISORDERS
●POOR BALANCE (ATAXIA)
TREMOR
● HEADACHE

**PERIPHERAL NERVOUS SYSTEM**
●WEAKNESS
SENSORY ABNORMALITIES

**MUSCULOSKELETAL**
●JOINT STIFFNESS OR CREPITANCE
●JOINT ACHING OR SWELLING
MUSCULAR PAINS OR CRAMPS

**SKIN**
RASH
ITCHING OR BURNING
HIVES OR BULLAE
SKIN INFECTIONS
OTHER SKIN LESIONS

**ENDOCRINE**
HORMONAL DYSFUNCTION

**IMMUNE SYSTEM**
IMMUNE DYSFUNCTION

**GENERAL**
GENERALIZED WEAKNESS
DECREASED STAMINA
FATIGUE

APPENDIX L-2

# ARNOLD SOMMERS

AGE 42
LIFE EXPECTANCY 36.02

SYMPTOMS

E
- ●WATERING
- BLURRED VISION
- ●RED, IRRITATED, ETC.

- DISCOMFORT OR PAIN
- TINNITIS
- ● DECREASED ACUITY

SE
- ●NOSE BLEEDS
- ●NASAL CONGESTION
- ●IRRITATED NOSE
- SNEEZING

ROAT
- ●SORENESS OR DIFFICULTY SWALLOWING

LMONARY
- ●SHORTNESS OF BREATH
- ●COUGH
- WHEEZE
- CHEST HEAVINESS
- HEMOPTYSIS

DIOVASCULAR
- PERIPHERAL VASCULAR
- CHEST PAIN
- ●ARRHYTHMIA

TROINTESTINAL
- GASTROINTESTINAL DISTRESS
- ●VOMITING
- ●NAUSEA
- HEMORRHOIDS AND RECTAL BLEEDING
- CONSTIPATION
- DIARRHEA
- ANOREXIA
- WEIGHT LOSS

ITOURINARY
- POOR CONTROL
- PAIN OR DIFFICULTY IN URINATION
- FREQUENCY
- NOCTURIA

REPRODUCTIVE OR SEXUAL DYSFUNCTION
- DECREASED LIBIDO
- IMPOTENCE, MALE
- MENSTRUAL DISORDERS, FEMALE

CENTRAL NERVOUS SYSTEM
- ●DIZZINESS OR LOSS OF CONSCIOUSNESS
- DECREASED CONCENTRATION OR MEMORY LOSS
- DEPRESSION
- ●NERVOUSNESS
- SLEEP DISORDERS
- POOR BALANCE (ATAXIA)
- ●TREMOR
- ●HEADACHE

PERIPHERAL NERVOUS SYSTEM
- WEAKNESS
- ●SENSORY ABNORMALITIES

MUSCULOSKELETAL
- JOINT STIFFNESS OR CREPITANCE
- JOINT ACHING OR SWELLING
- ●MUSCULAR PAINS OR CRAMPS

SKIN
- RASH
- ●ITCHING OR BURNING
- HIVES OR BULLAE
- ●SKIN INFECTIONS
- OTHER SKIN LESIONS

ENDOCRINE
- HORMONAL DYSFUNCTION

IMMUNE SYSTEM
- IMMUNE DYSFUNCTION

GENERAL
- GENERALIZED WEAKNESS
- DECREASED STAMINA
- FATIGUE

APPENDIX L-3

# LANCE TURLEY

AGE 22
LIFE EXPECTANCY 55.16

SYMPTOMS

E
- ● WATERING
- BLURRED VISION
- RED, IRRITATED, ETC.

R
- DISCOMFORT OR PAIN
- TINNITIS
- DECREASED ACUITY

SE
- ● NOSE BLEEDS
- ● NASAL CONGESTION
- ● IRRITATED NOSE
- SNEEZING

ROAT
- ● SORENESS OR DIFFICULTY SWALLOWING

LMONARY
- SHORTNESS OF BREATH
- ● COUGH
- WHEEZE
- ● CHEST HEAVINESS
- HEMOPTYSIS

RDIOVASCULAR
- PERIPHERAL VASCULAR
- CHEST PAIN
- ARRHYTHMIA

STROINTESTINAL
- GASTROINTESTINAL DISTRESS
- VOMITING
- NAUSEA
- HEMORRHOIDS AND RECTAL BLEEDING
- CONSTIPATION
- DIARRHEA
- ANOREXIA
- WEIGHT LOSS

ITOURINARY
- POOR CONTROL
- PAIN OR DIFFICULTY IN URINATION
- FREQUENCY
- NOCTURIA

REPRODUCTIVE OR SEXUAL DYSFUNCTION
- DECREASED LIBIDO
- IMPOTENCE, MALE
- MENSTRUAL DISORDERS, FEMALE

CENTRAL NERVOUS SYSTEM
- DIZZINESS OR LOSS OF CONSCIOUSNESS
- ● DECREASED CONCENTRATION OR MEMORY LOSS
- DEPRESSION
- NERVOUSNESS
- SLEEP DISORDERS
- POOR BALANCE (ATAXIA)
- TREMOR
- ● HEADACHE

PERIPHERAL NERVOUS SYSTEM
- WEAKNESS
- ● SENSORY ABNORMALITIES

MUSCULOSKELETAL
- JOINT STIFFNESS OR CREPITANCE
- ● JOINT ACHING OR SWELLING
- MUSCULAR PAINS OR CRAMPS

SKIN
- ● RASH
- ● ITCHING OR BURNING
- ● HIVES OR BULLAE
- SKIN INFECTIONS
- OTHER SKIN LESIONS

ENDOCRINE
- HORMONAL DYSFUNCTION

IMMUNE SYSTEM
- IMMUNE DYSFUNCTION

GENERAL
- GENERALIZED WEAKNESS
- ● DECREASED STAMINA
- ● FATIGUE

# DOROTHY BRADLEY

PHYSICAL FINDINGS

**EYE**

●CONJUNCTIVITIS

**EAR**

CHRONIC EAR INFECTION

**NOSE**

●MUCOUS MEMBRANE IRRITATION

**THROAT**

REDNESS

**PULMONARY**

ABNORMALITY OF LUNGS

**CARDIOVASCULAR**

DIMINISHED PERIPHERAL PULSE
OTHER PERIPHERAL VASCULAR ABNORMALITY
ABNORMAL HEART SOUNDS OR RHYTHM
ABNORMAL PULSE RATE

**GASTROINTESTINAL**

HEMORRHOIDS
LIVER AND OTHER ABDOMINAL ORGANS

**CENTRAL NERVOUS SYSTEM**

●CEREBELLUM
EIGHTH NERVE
●INCREASED DEEP TENDON REFLEXES
CRANIAL NERVES (EXCLUDING EIGHTH)

**PERIPHERAL NERVOUS SYSTEM**

●SENSORY
DECREASED OR ABSENT DEEP TENDON REFLEXES

**ENDOCRINE**

BREAST ABNORMALITY

# ARNOLD SOMMERS

## PHYSICAL FINDINGS

### EYE
- ● CONJUNCTIVITIS

### EAR
- CHRONIC EAR INFECTION

### NOSE
- ● MUCOUS MEMBRANE IRRITATION

### THROAT
- ● REDNESS

### PULMONARY
- ABNORMALITY OF LUNGS

### CARDIOVASCULAR
- DIMINISHED PERIPHERAL PULSE
- OTHER PERIPHERAL VASCULAR ABNORMALITY
- ABNORMAL HEART SOUNDS OR RHYTHM
- ABNORMAL PULSE RATE

### GASTROINTESTINAL
- HEMORRHOIDS
- LIVER AND OTHER ABDOMINAL ORGANS

### CENTRAL NERVOUS SYSTEM
- ● CEREBELLUM
- ● EIGHTH NERVE
- INCREASED DEEP TENDON REFLEXES
- CRANIAL NERVES (EXCLUDING EIGHTH)

### PERIPHERAL NERVOUS SYSTEM
- ● SENSORY
- DECREASED OR ABSENT DEEP TENDON REFLEXES

### ENDOCRINE
- BREAST ABNORMALITY

# LANCE TURLEY

## PHYSICAL FINDINGS

### EYE
- CONJUNCTIVITIS

### EAR
CHRONIC EAR INFECTION

### NOSE
- MUCOUS MEMBRANE IRRITATION

### THROAT
REDNESS

### PULMONARY
ABNORMALITY OF LUNGS

### CARDIOVASCULAR
DIMINISHED PERIPHERAL PULSE
OTHER PERIPHERAL VASCULAR ABNORMALITY
ABNORMAL HEART SOUNDS OR RHYTHM
ABNORMAL PULSE RATE

### GASTROINTESTINAL
HEMORRHOIDS
LIVER AND OTHER ABDOMINAL ORGANS

### CENTRAL NERVOUS SYSTEM
CEREBELLUM
EIGHTH NERVE
INCREASED DEEP TENDON REFLEXES
CRANIAL NERVES (EXCLUDING EIGHTH)

### PERIPHERAL NERVOUS SYSTEM
SENSORY
DECREASED OR ABSENT DEEP TENDON REFLEXES

### ENDOCRINE
BREAST ABNORMALITY

# DOROTHY BRADLEY

LABORATORY

R

●● 500 FREQUENCY
●● 1000 FREQUENCY
●● 2000 FREQUENCY
●● 3000 FREQUENCY
●● 4000 FREQUENCY
●● 6000 FREQUENCY

ULMONARY

● FVC
$FEV_1$/FVC
CHEST X-RAY

ARDIOVASCULAR

EKG

IVER METABOLISM

PORPHYRINS
UROPORPHYRIN
● HEPTACARBOXYLIC
PENTACARBOXYLIC
PORPHOBILINOGEN
COPROPORPHYRIN ● RATIO
ENZYMES
GGTP
SGOT
SGPT
ALKALINE PHOSPHATASE
● LDH
● IRON METABOLISM
PROTHROMBIN TIME
LIPID METABOLISM
HDL CHOLESTEROL
LDL CHOLESTEROL
CHOLESTEROL
TRIGLYCERIDES
LIPOPROTEIN PHENOTYPE
PROTEIN METABOLISM
TOTAL PROTEIN (ELECTROPHORESIS)
TOTAL PROTEIN (SMAC)
A/G
ALPHA 2 GLOBULIN
GAMMA GLOBULIN

NITOURINARY

URINALYSIS
● BUN
CREATININE
● URIC ACID
CALCIUM AND PHOSPHORUS
● POTASSIUM
CHLORIDE

MIDWEST IMMUNE HEMA

● ●
● ●
● ●

FERTILITY ANALYSIS

GRADE
TYPE MOTION
PERCENT ACTIVITY
COUNT
MORPHOLOGY

CENTRAL NERVOUS SYSTEM

ZUNG ANXIETY SCALE
● NEUROBEHAVIORAL BATTERY
BECK DEPRESSION INVENTORY

PERIPHERAL NERVOUS SYSTEM

EMG
PNVC, MOTOR
PNVC, SENSORY

MUSCULOSKELETAL

CPK

ENDOCRINE

THYROID
T3 UPTAKE
T4 TSH RIA
FT Index
GLUCOSE
TESTOSTERONE

IMMUNE SYSTEM

IMMUNOGLOBULIN
G
A
M
MITOGEN CHALLENGE
UNSTIMULATED CPM
PHA CPM
PHA STIMULATION INDEX
CON A CPM
CON A STIMULATION INDEX
PWM CPM
PWM INDEX
WHITE BLOOD CELLS
TOTAL LYMPHOCYTES (%)
T LYMPHOCYTES (%)
EOSINOPHIL
SEGS
MONOCYTES
● BASOPHILS
TOTAL

# ARNOLD SOMMERS

LABORATORY

APPENDIX N-2

AR
- 500 FREQUENCY
- 1000 FREQUENCY
- 2000 FREQUENCY
- ●3000 FREQUENCY
- ●●4000 FREQUENCY
- ●●6000 FREQUENCY

LMONARY
- FVC
- $FEV_1$/FVC
- CHEST X-RAY

RDIOVASCULAR
- EKG

VER METABOLISM
- PORPHYRINS
  - UROPORPHYRIN
  - HEPTACARBOXYLIC
  - PENTACARBOXYLIC
  - ●PORPHOBILINOGEN
  - ●COPROPORPHYRIN ●RATIO
- ENZYMES
  - ●GGTP
  - ●SGOT
  - ●SGPT
  - ●ALKALINE PHOSPHATASE
  - ●LDH
- IRON METABOLISM
- PROTHROMBIN TIME
- LIPID METABOLISM
  - HDL CHOLESTEROL
  - ●LDL CHOLESTEROL
  - ●CHOLESTEROL
  - ●TRIGLYCERIDES
  - ●LIPOPROTEIN PHENOTYPE
- PROTEIN METABOLISM
  - TOTAL PROTEIN (ELECTROPHORESIS)
  - TOTAL PROTEIN (SMAC)
  - A/G
  - ALPHA 2 GLOBULIN
  - GAMMA GLOBULIN

ITOURINARY
- URINALYSIS
- BUN
- ●CREATININE
- URIC ACID
- CALCIUM AND PHOSPHORUS
- POTASSIUM
- CHLORIDE

MIDWEST

IMMUNE ●●

HEMA ●●●●

**FERTILITY ANALYSIS**
- GRADE
- TYPE MOTION
- PERCENT ACTIVITY
- COUNT
- MORPHOLOGY

**CENTRAL NERVOUS SYSTEM**
- ZUNG ANXIETY SCALE
- ●NEUROBEHAVIORAL BATTERY
- BECK DEPRESSION INVENTORY

**PERIPHERAL NERVOUS SYSTEM**
- ●EMG
- ●PNVC, MOTOR
- ●PNVC, SENSORY

**MUSCULOSKELETAL**
- CPK

**ENDOCRINE**
- THYROID
  - T3 UPTAKE
  - T4 TSH RIA
  - FT Index
- GLUCOSE
- TESTOSTERONE

**IMMUNE SYSTEM**
- IMMUNOGLOBULIN
  - G
  - A
  - M
- MITOGEN CHALLENGE
  - UNSTIMULATED CPM
  - PHA CPM
  - PHA STIMULATION INDEX
  - CON A CPM
  - CON A STIMULATION INDEX
  - PWM CPM
  - PWM INDEX
- WHITE BLOOD CELLS
  - TOTAL LYMPHOCYTES (%)
  - T LYMPHOCYTES (%)
  - ●EOSINOPHIL
  - SEGS
  - MONOCYTES
  - BASOPHILS
  - TOTAL

# ANCE TURLEY

APPENDIX N-3

LABORATORY

500 FREQUENCY
1000 FREQUENCY
2000 FREQUENCY
3000 FREQUENCY
4000 FREQUENCY
6000 FREQUENCY

ONARY
FVC
$FEV_1$/FVC

CHEST X-RAY

IOVASCULAR
EKG

R METABOLISM
PORPHYRINS
UROPORPHYRIN
HEPTACARBOXYLIC
PENTACARBOXYLIC
PORPHOBILINOGEN
●COPROPORPHYRIN RATIO
ENZYMES
GGTP
SGOT
SGPT
ALKALINE PHOSPHATASE
LDH
IRON METABOLISM
PROTHROMBIN TIME
LIPID METABOLISM
HDL CHOLESTEROL
LDL CHOLESTEROL
CHOLESTEROL
TRIGLYCERIDES
LIPOPROTEIN PHENOTYPE
PROTEIN METABOLISM
TOTAL PROTEIN (ELECTROPHORESIS)
TOTAL PROTEIN (SMAC)
A/G
ALPHA 2 GLOBULIN
GAMMA GLOBULIN
OURINARY
URINALYSIS
BUN
CREATININE
URIC ACID
CALCIUM AND PHOSPHORUS
POTASSIUM
CHLORIDE

MIDWEST IMMUNE HEMA

FERTILITY ANALYSIS
●GRADE
●TYPE MOTION
●PERCENT ACTIVITY
●COUNT
●MORPHOLOGY

CENTRAL NERVOUS SYSTEM
ZUNG ANXIETY SCALE
NEUROBEHAVIORAL BATTERY
BECK DEPRESSION INVENTORY

PERIPHERAL NERVOUS SYSTEM
EMG
PNVC, MOTOR
PNVC, SENSORY

MUSCULOSKELETAL
CPK

ENDOCRINE
THYROID
T3 UPTAKE
T4 TSH RIA
FT Index
GLUCOSE
TESTOSTERONE

IMMUNE SYSTEM
IMMUNOGLOBULIN
G
A
● M
MITOGEN CHALLENGE
UNSTIMULATED CPM
PHA CPM
PHA STIMULATION INDEX
CON A CPM
CON A STIMULATION INDEX
PWM CPM
PWM INDEX
WHITE BLOOD CELLS
●TOTAL LYMPHOCYTES(%)
T LYMPHOCYTES (%)
EOSINOPHIL
●SEGS
MONOCYTES
BASOPHILS
●TOTAL

DOROTHY BRADLEY

DIAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) Conjunctival damage with hemorrhage
2) Bilateral hearing loss with 8th cranial nerve damage
3) Chronic rhinitis
4) Liver dysfunction including:
    a) abnormal LDH
    b) abnormal iron metabolism
    c) abnormal porphyrin metabolism
5) Early renal insufficiency
6) Central nervous system dysfunction including:
    a) cerebeller damage
    b) diffuse cerebral damage
7) Peripheral nervous system dysfunction
8) Immune system dysfunction

PROGNOSIS: VERY GUARDED

RNOLD SOMMERS

IAGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) Chronic conjunctivitis.
2) Bilateral neurosensory hearing loss with 8th cranial nerve damage.
3) Chronic rhinitis and pharyngitis with nasal hemorrhage.
4) Chronic chemical bronchitis.
5) Acute recurrent gastritis (possible porphyria crises).
6) Liver dysfunction, including:
   a) Severe chemical hepatitis.
   b) Abnormal lipid metabolism.
   c) Abnormal porphyrin metabolism.
7) Genitourinary tract dysfunction with probable renal insufficiency.
8) Central nervous system dysfunction, including:
   a) Cerebral damage.
   b) Probable cerebellar damage.
9) Peripheral polyneuropathy.
10) Musculoskeletal dysfunction.
11) Immune system dysfunction.
12) Hematologic system dysfunction.

ROGNOSIS: POOR TO VERY GUARDED

# APPENDIX O-3

LLIAM LANCE TURLEY

AGNOSIS: Chronic Systemic Chemical Intoxication caused by the Alcolac toxic chemicals with damage to multiple organ systems, including:

1) Chronic bilateral conjunctivitis

2) Chronic rhinitis and pharyngitis with recurrent bleeding

3) Liver dysfunction including:

   a) abnormal porphyrin metabolism

   b) abnormal prothrombin time

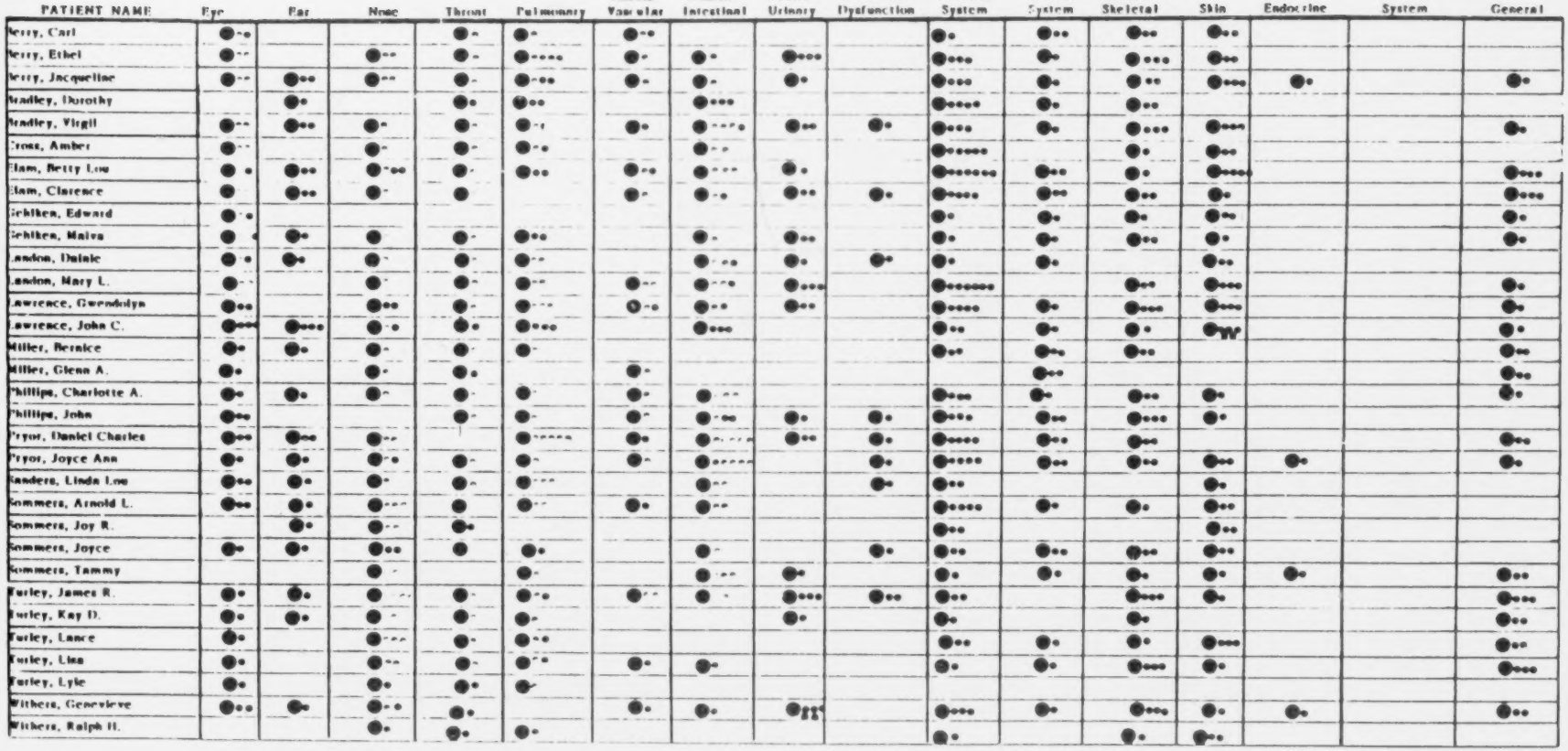
4) Reproductive system dysfunction

5) Chronic chemical dermatitis with probable hypersensitivity component

6) Immune system dysfunction with depression

OGNOSIS: VERY GUARDED

# SUMMARY OF SYMPTOMS




APPENDIX P-1

# SUMMARY OF ABNORMAL PHYSICAL FINDINGS

| PATIENT NAME | Eye | Ear | Nose | Throat | Pulmonary | Cardio-Vascular | Gastro-Intestinal | Central Nervous System | Peripheral Nervous System | Endocrine |
|---|---|---|---|---|---|---|---|---|---|---|
| Berry, Carl | ● | | ● | | | ● | | | | |
| Berry, Ethel | | | ● | | | ● | ● | | ●● | |
| Berry, Jacqueline | | ● | ● | | | | | ●● | | |
| Bradley, Dorothy | ● | | ● | | | | | ●● | ● | |
| Bradley, Virgil | | | ● | | | | | ● | ● | |
| Cross, Amber | ● | | | ● | | | ● | | | |
| Elam, Betty Lou | | | ● | | ● | ●● | | ● | | |
| Elam, Clarence | | | ● | | | | | | | |
| Gehlken, Edward | | | | | | | | | | |
| Gehlken, Malva | | | ● | ● | | | ● | ● | ●● | ● |
| Landon, Dainie | | | ● | | ● | ●● | ● | ●● | ●● | |
| Landon, Mary L. | | | | | | ● | | ● | | |
| Lawrence, Gwendolyn | | | | | ● | ● | | ●● | | |
| Lawrence, John C. | | | ● | | ● | ● | ● | | ● | |
| Miller, Bernice | ● | | ● | | | | | ● | ● | |
| Miller, Glenn A. | ● | | ● | | | | | ● | ● | |
| Phillips, Charlotte A. | | | | | | ●● | | | ● | |
| Phillips, John | | ● | | | ● | ● | ● | ●●● | ● | |
| Pryor, Daniel Charles | | | ● | | ● | ● | | | | |
| Pryor, Joyce Ann | ● | ● | ● | | | ●● | ● | ●● | | ● |
| Sanders, Linda Lou | | | | | | | | | | |
| Sommers, Arnold L. | ● | | ● | ● | | | | ●● | ● | |
| Sommers, Joy R. | | | | | | | | | ● | |
| Sommers, Joyce | ● | | ● | | | ● | | ● | | ● |
| Sommers, Tammy | | | ● | | | | | | | |
| Turley, James R. | | | | | | | | | ● | |
| Turley, Kay D. | | | ● | | | | | | | |
| Turley, Lance | ● | | ● | | | | | | | |
| Turley, Lisa | | | ● | | | ● | | | ● | |
| Turley, Lyle | | ● | | | | | | | ● | |
| Withers, Genevieve | | | ● | | | ● | | ● | | |
| Withers, Ralph H. | | | ● | | | | ● | ● | | |

# SUMMARY OF ABNORMAL LABORATORY TESTS

July Mid-Aug '85 West '84

| PATIENT NAME | Ear | Pulmonary | Cardio-Vascular | Liver Metabolism | Genito-Urinary | Fertility Analysis | Central Nervous | Periph. Nervous | Musculo-Skeletal | Endocrine | Immune System |
|---|---|---|---|---|---|---|---|---|---|---|---|
| erry, Carl | ● | | | ● | | ● | ● | | ● | | ● |
| erry, Ethel | ● | ● | | ● | ● | | ● | | | ● | ● |
| erry, Jacqueline | ● | | | ● | ● | | ● | | ● | ● | ● |
| radley, Dorothy | ● | ● | | ● | ● | | ● | | | | ● |
| radley, Virgil | ● | | | ● | ● | ● | ● | ● | | ● | ● |
| ross, Amber | | | | ● | ● | | ● | | | ● | ● |
| lam, Betty Lou | ● | | | ● | ● | | ● | ● | | | ● |
| lam, Clarence | ● | | | ● | ● | Vasectomy | | ● | | | ★ |
| Schiken, Edward | | ✗ | ✗ | ✗ | ✗ | ✗ | ✗ | | | | |
| Schiken, Malva | ● | | | ● | | | | ● | | ● | ● |
| Landon, Dainie | ● | ● | ● | ● | ● | ★ | | | | ● | ● |
| Landon, Mary L. | ● | | | ● | ● | | ● | ● | | ★ | ● |
| Lawrence, Gwendolyn | | | | ● | ● | | ● | ● | ● | ● | ● |
| Lawrence, John C. | ● | | | ● | | ● | | | | | ● |
| Miller, Bernice | ● | | | ● | ● | | | | | ● | ● |
| Miller, Glenn A. | ● | | ● | ● | ● | ● | ● | | | | ● |
| Phillips, Charlotte A. | ● | | | ● | ● | | ● | | | ● | ● |
| Phillips, John | ● | ● | | ● | ● | ★ | ● | ● | | | ● |
| Pryor, Daniel Charles | | ● | | ● | | ● | ● | | | ● | ● |
| Pryor, Joyce Ann | | | | ● | ● | | ● | | | ● | ● |
| Sanders, Linda Lou | ● | | | ● | | | ● | | | | ● |
| Sommers, Arnold L. | ● | | | ● | ● | ✗ | ● | ● | | | ● |
| Sommers, Joy R. | | | | ● | ● | | | | | | ● |
| Sommers, Joyce | ● | | | ● | ● | | ● | | | ● | ● |
| Sommers, Tammy | | | | ● | | | ● | | | ● | ● |
| Turley, James R. | ● | | | ● | ● | ★ | ● | ● | | | ● |
| Turley, Kay D. | | | | ● | | | ● | ● | | ● | ● |
| Turley, Lance | | | ★ | ● | ● | ● | | | | | ● |
| Turley, Lisa | | | | ● | ● | | ● | | | ● | ● |
| Turley, Lyle | | ✗ | | ● | ● | | | | | | ● |
| Withers, Genevieve | | | | ● | | | | | | ● | ● |
| Withers, Ralph H. | ● | ● | ● | ● | ● | ● | | | | | ● |

| DOROTHY BRADLEY | ALCOLAC | ANOTHER CAUSE | PRE-EXISTING | UNKNOWN |
|---|---|---|---|---|
| Recurrent eye irritation by history. | ● | | | |
| Mild hearing loss — presbycusis. | | ● | | |
| Mild to moderate dementia — secondary to hypertension probably. | | ● | | |
| Osteoarthritis right knee. | | ● | | |
| Hypertension. | | ● | ● | |
| Obesity. | | ● | ● | |
| Cholecystectomy-appendectomy-1970 | | ● | ● | |
| Partial breast resection-1978 | | ● | ● | |

| ARNOLD SOMMERS | ALCOLAC | ANOTHER CAUSE | PRE-EXISTING | UNKNOWN |
|---|---|---|---|---|
| ...istory of eye, nose and throat irritation. | ● | | | |
| ...hronic bronchitis — secondary to smoking. | | ● | | |
| ...robable alcohol-induced fatty liver ...enzyme and lipid type pattern). | | ● | | |
| ...laralgia paresthetica. | | ● | | |
| ...ccupational noise-induced hearing loss. | | ● | | |
| ...istory of duodenal ulcer. | | ● | ● | |

APPENDIX G-3

| LANCE TURLEY | ALCOLAC | ANOTHER CAUSE | PRE-EXISTING | UNKNOWN |
|---|---|---|---|---|
| History of nasal irritation and nosebleed. | ● | | | |
| S/P surgery to knee following injury in 1980. | | ● | | |